ARCHIVES OF OPHTHALMOLOGY.

A SUCCESSFULLY OPERATED CASE OF SUPRA-SELLAR CYST.[1]

BY DR. HARRY S. GRADLE, CHICAGO.

(With six figures in the text.)

THE patient, a man of 33 years of age, was first seen on October 17, 1924, with the complaint that he had had difficulty in reading for the past year, which had been increasing greatly during the past six weeks. At first the right eye had been the more involved; but of recent weeks, the left vision had been the more unsatisfactory. He was neither a smoker nor drinker. His general physical condition had always been normal and there had been no change in his appearance or weight for some time past. No other items in the amnesis were of account, except for fronto-occipital headaches of irregular character and of long standing.

Upon examination, he was found to have less than 0.1 vision in either eye. The eyes were externally normal and showed no limitation of motion. The pupils were 6*mm* in diameter, equal in size, and rigid. Ophthalmoscopically, the disks were clear cut and normal. The retinal veins were possibly slightly dilated. The visual fields (vide I) were very markedly contracted, but not in diagnostic form, although the left field was extremely suggestive of interference with transmission by the fibers in the upper half of the nerve. The patient was seen by Dr. Lewis Pollack who reported a negative neurological examination, apart from the eye findings.

October 24, 1924. The left vision had decreased to such

[1] Read before the Chicago Neurological Society, April 15, 1926.

an extent that it was not possible to obtain the visual field of the left eye.

November 13, 1924. Physical examination revealed no abnormalities. Blood and spinal Wassermann negative. Roentgenograms of head showed no abnormalities. After having been flat upon his back in bed for a week, the vision in the left eye improved up to finger counting. During this period, a visual field taken with the fingers suggested a bi-temporal hemianopsia, but the vision was too poor to admit of corroboration with the perimeter. The right visual field varied slightly from day to day. A tentative diagnosis of supra-sellar cyst was made.

November 14, 1924. Dr. George Davenport made a large left temporo-frontal flap and an underlying bone flap about four inches in diameter. The dura was incised and the brain found to be very wet. The chiasm was exposed and a supra-sellar cyst found. About 20*cc* of a lemon-colored fluid were evacuated and the superior and anterior wall of the cyst torn away. Slight bleeding from the middle meningeal artery was easily stopped. The posterior and inferior walls and the pedicle could not be seen.

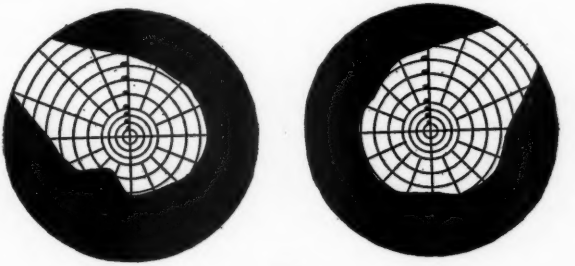
November 15, 1924. Patient in good condition and reports improved vision.

December 20, 1924. The recovery from the operation was uneventful. Both pupils now dilated ad maximum and rigid. There is a definite pallor of both optic disks. R. V. $\frac{1}{100}$; L. V. 0.3. Visual fields (vide II). The improvement from here on was slow.

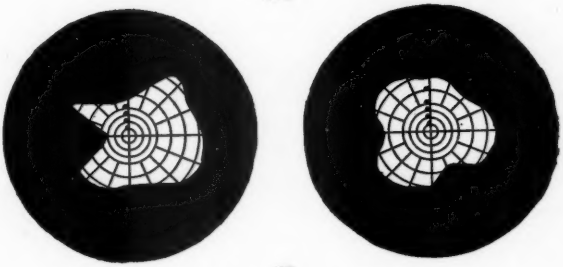
December 31, 1925. The pupils are smaller and respond slowly to light and accommodation. The atrophy of both disks progressed slowly for about six months, but has been stationary as far as can be detected for the past six months. R. V. 0.3.; L. V. 0.2 +. Combined 0.4. Visual fields (vide III). The patient has been carrying on his former occupation as Superintendent of Schools with hindrance and without symptoms.

The most interesting features of the case are those presented by the visual fields. In Field I, the left field is very suggestive of pressure upon the superior aspect of the left optic nerve, anterior to the chiasm and posterior to the entrance of the vessels into the optic nerve. The involvement of the right field in Field I is not in any way diagnostic per se; but when taken in connection with the left field, indicates that the process involved the right optic nerve as well as the

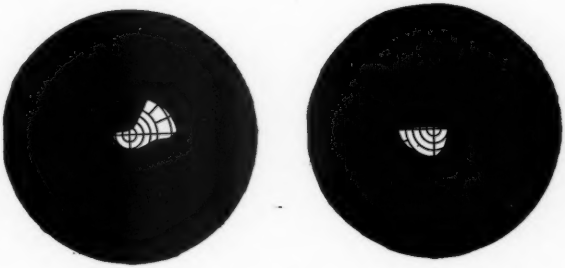
left. Consequently, the process, whatever it was, must of necessity be located where the right and left optic nerves



III



II



I

joined, namely, at the anterior border of the optic chiasm. Later another field suggests that the process had increased in size and was leaning over toward the left, completely blocking nerve impulses through the left optic nerve. At the same

time, the superior portion of the right optic nerve was somewhat more viable, but the tendency toward a hemianopsia indicated pressure on the inner aspect of the right nerve, anterior to the chiasm. One week later, a finger field showed a definite bi-temporal hemianopsia; this was after one week in bed. Reasoning thus from the fields and the lack of other findings, the process had to be one that involved the anterior portion of the optic chiasm, probably superiorly, and was of a character that changed its location somewhat with growth and with the posture of the patient. The only process fulfilling these requirements was a supra-sellar cyst, as it proved to be at operation.

CONGENITAL TUMOR MASS AT THE PERIPHERY OF THE OPTIC DISK.

By Dr. ARTHUR M. YUDKIN, New Haven.

From the Department of Surgery, Section of Ophthalmology, Yale University School of Medicine.

(*With one illustration on Text-Plate XXIII.*)

ABNORMALITIES of the intraocular portion of the optic nerve and its sheaths, except for colobomata are rare. Most of the cases of tumor masses about the optic nerve which have been reported in the literature are of the malignant type. In view of the rarity of benign growths in this area, I report the following case.

Miss R. P., 14 years old, was first seen by me December 17, 1923. She complained of headaches which were present after doing close work. There was no family history of ocular disturbance. The patient was well until two years ago, when she suffered from a post diphtheritic laryngitis, but has had no ocular trouble as long as she can remember.

Ocular examination: Vision O.D. $\frac{10}{10}$
O. S. $\frac{10}{10}$

Peripheral field normal and ocular tension normal in both eyes. The examination of the right eye showed nothing abnormal. The left eye presented the following fundus picture. The fundus pigmentation was the same as in the right eye, the optic nerve outline was well defined but in the upper three quarters of the nasal side of the disk was a raised, almost circular pearly white mass. The elevation was approximately 2 diopters above the fundus, and about one and one-half disk diameters in its longest direction. The margin of this tissue was well defined, showing little, if

any, pigment in its structure. The area covering the disk margin seemed to be part of the lip of the excavation of the optic nerve. The retinal and choroidal tissue surrounding this mass showed no pathological changes. In the lower nasal part of the elevation was a peculiar inverted V area of pigment which resembled the hooklets of a cysticercus, but there was nothing else to substantiate this finding. A large blood vessel crossed the tissue in an undulating manner, giving the impression of detachment of the retina by a semi solid or cystic mass. There were several other small vessels on the elevation. No other abnormal processes were noted at this examination.

The patient was requested to return for further examination but did not do so.

In January, 1924, I sent for her and after examination, found that the fundus picture had not changed and the mass was the same size, visual acuity and peripheral vision were normal. Patient was seen in July and again in December of 1925. At this time, a complete physical examination was made. The suspicious laryngitis was investigated by Dr. Swain who found nothing abnormal in the larynx and attributed the hoarseness to the old diphtheritic condition. Blood was taken for Wassermann, a tuberculin test was made, and urine examined, but all were negative.

The blind spot as may be seen in the chart was increased. Pressure on the eyeball showed no pulsation of the vessels on the mass. A close examination of the vitreous showed two delicate clumps of tissue which were quite forward but were fairly stationary when the eye was moved.

I considered the larger of the two as a remnant of Cloquet's canal though no signs of hyaloid artery on the posterior lens capsule was to be seen even with the slit-lamp. There were no other floating bodies in the vitreous and no suggestion of any inflammatory processes present.

COMMENT.

I am inclined to consider this mass a congenital anomaly which is intimately associated with an obliterated hyaloid system. A thorough search of the literature reveals very little of a similar nature. (1) De Beck reports cases from his own

ILLUSTRATING DR. ARTHUR M. YUDKIN'S ARTICLE, "CONGENITAL TUMOR MASS AT THE PERIPHERY OF THE OPTIC DISK."

experience and that of others which have some resemblance, in fact, to my case. The greatest difference is that in his description the tumor mass covers the blood vessels. (2) Randall and (3) Parsons describe fundus changes of a similar character. The outstanding features of this case are (1) the normal visual acuity and peripheral field of vision; (2) the circumscribed and enlarged blind spot; (3) the fundus shows no other pathological disturbance; (4) the area under observation has not changed in over two years; and (5) there are other remnants of the hyaloid system in the vitreous cavity.

BIBLIOGRAPHY.

1. De Beck, D. *Amer. Ophth. Monographs*, vol. iii, p. 1, 1890.
2. Randall, B. A. *Trans. of Amer. Ophth. Soc.*, vol. v, p. 117, 1888.
3. Parsons. *The Pathology of the Eye*, vol. iii, p. 851, 1906.

SUSPENSION OF THE FUSION IMPULSE AS A THERAPEUTIC MEASURE.[1]

By Dr. ELLICE M. ALGER, New York.

WE have been taught of late years to consider binocular single vision as of superlative importance. Tasks involving estimates of relief and distance and solidity are said to be impossible without it. Its loss from industrial accident or disease is considered a special factor in the compensation award. Its congenital weakness or absence is said to be the fundamental cause of strabismus: the cause rather than the result of an amblyopia. It is urged as a prerequisite for a career in the army or navy or, in civil life, as a chauffeur. Biologically it is said to be one of the chief factors in the very evolution of man.

Most people maintain binocular single vision with perfect ease. We see, however, many who have never had it or who have lost it but who seem to get along for all practical purposes as well as their fellows, their defect often being discovered only by accident.

We see others who have what might be called partial binocular single vision, some of them having it for distance and not for near work and others vice versa. All these people are notoriously exempt from the troublesome set of symptoms which we group together as muscular asthenopia. Even those who lose their fusion power suddenly from accident or disease are much more annoyed by the faulty projection than by the diplopia. But there is another group of patients who have a strong and imperious fusion impulse which by reason of some neuro muscular spasm or insufficiency they are able to gratify only with the greatest difficulty. We spend a large

[1] Read before Am. Ophth. Soc., Hot Springs, Va., June, 1926.

part of our time trying to help them by various means, with glasses, prisms, exercises or operations, but many of them flit from one ophthalmologist to another without much relief and lead useless and sometimes very unhappy lives.

I have been very much impressed over a period of years by the rather remarkable relief obtained in occasional cases by a frank abandonment of attempts to maintain binocular single vision and have been led to speculate:

(1) Whether the practical value of binocular single vision, for many people at least, has not been entirely over-estimated on grounds that are largely theoretical.

(2) Whether in many cases, as in strabismus for instance, we are doing our patients any real kindness by trying to develop a taste for binocular single vision for which they have neither the fundamental impulse nor the necessary coördination.

(3) Whether in some individuals who are already maintaining binocular vision at too great a cost it might not be better to suspend the fusion impulse, wholly or in part.

(4) Some practical methods.

Most of our knowledge of binocular vision has resulted from the acute observations of physiologists and psychologists on their own sensations and those of their students, that is to say on healthy normal individuals. Now binocular single vision is not a mere mixture of retinal sensations but a series of judgments based on those sensations, in which even in ordinary people, experience and perhaps imagination play a large part. The studied judgments of a trained psychologist might be quite different from the instinctive ones of the ordinary individual, just as his are quite certainly different from those of the patient who has been born without some of the essential factors. We are apt to assume that because we are badly confused when we bandage one eye that the one-eyed man is equally at loss. We are apt to assume that because the strabismic suppresses one image that he sees things as a one-eyed man would, which is far from being true. In other words the whole subject has been considered from the theoretical laboratory point of view rather than from the basis of practical every day life.

Without question most normal individuals base their ideas

of size and distance and solidity and the like chiefly on stereoscopic vision. Most of the laboratory experiments are quite properly purposely so constructed as to exclude all other factors. But in real life we are dealing for the most part with situations that are fairly familiar and in which we have many facts that unconsciously influence our judgments, such as light and shade, the comparison with other objects of which we know both form and size, small movements of the eye or head and the use of our other senses. It is quite likely too, that people who have had to rely on these other factors exclusively acquire much greater skill in their use than is possible to the rest of us. Certainly no one who has watched birds catch insects in the air, or weave their way through a thicket or unerringly light on a twig can doubt that there are other ways of estimating distance or localizing objects in space than by stereoscopic vision. Certainly we see every day people without binocular single vision earning a competitive living by doing things that should be difficult or impossible for them. The strabismic finds his defect no bar to employment as mechanic. We find him playing with reasonable success games like golf and tennis and baseball. Most of the work of the rhinologist and otologist is done with monocular vision only, and we have all known men who stood well in general surgery and even in ophthalmology who actually had only one eye.

We have very little actual information as to how far such men fall short of their fellows in actual practical tests nor of how they reach their visual judgments. A careful study of vision in a group of great batsmen or golfers or tennis players would be very well worth while, on the one hand, and on the other an equally careful study of our older strabismic patients as to their ability to play games, to paint pictures and model figures that have been supposed to require binocular single vision.

Binocular single vision is a complicated process, a chain in which we can recognize at least four distinct links, retinal stimulation, cerebral perception, neuro muscular coördination and a fusion impulse. We have to consider each of these in our attempts to build up binocular vision, and conversely we can attack any link in the chain in our effort to suspend it.

Stimulation.—If we are to fuse images it will be unquestionably easier if they are perfectly similar in size and shape and color and brightness. We secure this by careful correction of refraction and the use of test objects which shall give the maximum of retinal stimulation, and we can express its measure very much as we do ordinary central vision. But while fusion of images is built up around central vision it still survives to a degree without it as we find in our stereoscopic charts for mapping central scotomata.

Perception.—Every object in the field of vision causes some degree of retinal stimulation in the form of images more or less distinct. Some of them we see, others, we remember that we saw though we did not notice them at the time, others which must have been there we never did see. In other words there is a constant succession of impressions which we can ignore or bring into our consciousness at will. We can train ourselves to notice at once certain minute stimuli like a tubercle bacillus in a microscope field and to absolutely ignore more noticeable objects. The hysteric with his intense concentration on a fixed idea has great difficulty in bringing his retinal stimuli into his field of consciousness while the neurasthenic has a corresponding difficulty in keeping out the ones he does not want.

We constantly ignore retinal images in binocular vision just as we do in monocular. We know the position of our heads in space through our vestibular apparatus, of our eyes in our heads through our muscular sense and of objects in space by retinal projection and by focussing. In binocular vision we reason that images formed on "similar" points of each retina are fused and seen single while all others should be seen double. But it is easy to demonstrate that there are many images which could not possibly be superposed but which are not nevertheless seen double. In some cases the brain simply ignores one image or parts of both, entirely. In others it fuses them into a stereoscopic picture which is quite inconsistent with the retinal data. In strabismus for instance the brain pays attention to the stimuli of the squinting eye only so far as they do not conflict with those from the dominant fixing eye, but they can be brought into consciousness instantly by covering the fixing eye. This subordination pre-

vents the normal stereoscopic combinations, but the strabismic unless his deviation be very great has a nearly normal binocular field in which he projects and orients about as other people do. It has been argued that this subordination of one image must result eventually in an amblyopia exanopsia but it is difficult to see how a suppression which is purely psychic and in nature similar to what we are all constantly doing, could possibly have any such result. It seems to me there are several more plausible hypotheses to explain it.

The *Fusion Impulse* is a cerebral function of which we know relatively little. Some individuals have an antipathy to diplopia which compels them to attempt fusion to the very limit of their neuro muscular ability while others with just as good eyesight are either not conscious of diplopia at all or make but slight attempts at fusion. We have no very practical way of measuring the "fusion impulse" except by the patient's speed in fusing images which are made progressively dissimilar or less bright, when separated very slightly by weak prisms. We can also infer something from the heights of stereoscopic vision, though this involves other factors.

Fusion Power.—Movements of the eyeballs produced by the extrinsic muscles require almost no effort since the arc through which they have to move is very small. We can measure this as a simple muscular function by noting with the perimeter or the tropometer the rotations of the eyes in various directions or we can measure the actual muscular power developed. But the fusion movements of the eyeballs are not by any means so simple since they are accomplished by the coördination of groups of muscles in which the action of some is checked or assisted by the others. Every one who has ever played golf or tennis knows that coördination is more or less of a "knack" in which no two individuals are exactly alike, nor does the same individual coördinate as smoothly at one time as at another. Ideal coördination means a just sufficient stimulation of the primary muscles and no others, followed by the minimum stimulation of the secondary ones which check the first. Even the best athletes probably overstimulate more muscles than they really need, and have to apply unnecessary resistance from antagonists,

while most of us develop very little power from a vast expenditure of energy.

The analogy probably holds good for the eyes. Most of us fuse images with varying ease but many people keep their whole bunch of muscles resisting each other in a constant state of unnecessary cramp. We can measure the gross effect secured by the arc of movement made or by the amount of prism overcome, but this gives us not the slightest idea of the muscular power involved nor the nervous energy wasted in the process. If the forces were equally balanced the most extreme exertion might be evidenced only by a retraction of the globes. A strabismic converges his eyes far more than the ordinary esophoric patient but he is using much less muscular energy and suffers no particular strain or pain. If we can judge anything from the attempts to teach proper coördination of muscles in the various games the success of our efforts is in direct proportion to the youth of the student and his capacity for unconscious imitation of his teacher. Insofar as it is done consciously it is little better than useless.

If we should conclude that lack of fusion does not hopelessly handicap the average individual we must admit to ourselves that we have often used very poor judgment and wasted valuable time in our efforts to preserve or restore it.

It is perhaps fortunate that we can very seldom reëstablish fusion in our strabismus patients except in extreme youth and by the expenditure of time and patience that few of us have. Their very defect is evidence of weak impulse or poor coördination or both and if we improved the first without the second we should doubtless find that in many cases we had exchanged the comfortable more or less monocular vision of the strabismic for the inefficiency and discomfort of muscular asthenopia. Believing as I do that there is very little real evidence of amblyopia exanopsia I am much better satisfied with mere cosmetic results than I used to be.

It goes without saying that the fusion impulse should not be lightly suppressed but that every reasonable effort should be made especially in young patients to develop it and to make ocular coördination as easy and automatic as possible. Suppression should be reserved for those who have proved unequal to the strain of binocular single vision or for those

others whose age or occupation makes it of minor importance. We should also bear in mind the sharp distinction between fusion power and fusion impulse. The paralytic has lost his power but his impulse persists as strong as ever for a long time. The strabismic on the other hand often has an excess of power but no impulse whatever. It is the impulse we talk of suppressing. I have found it most useful.

(1) in some cases of marked anisometropia.
(2) after removal of cataract from both eyes.
(3) in some cases of incomplete motor paralysis.
(4) in some cases of insufficiency, especially of convergence.
(5) in some cases of motor spasm.

In such patients it allows the eyes to assume the position of rest with consequent relief of strain, or it frees the patient from the annoyance of diplopia—or it permits of the eyes being used for different focal distances. Many of you will remember hearing Prof. Gullstrand tell how he trained himself to disassociate his eyes so that he could wear a short focus glass over one eye and a longer one over the other for convenience in working.

We can accomplish our purpose in a variety of ways which must be adapted to the circumstances of the case. They naturally fall into these categories.

(1) those which absolutely exclude one eye.
(2) those which reduce the retinal stimulus.
(3) those which are primarily aimed at the fusion centers.

Exclusion Measures are among the oldest and most obvious means of abolishing the fusion impulse. Nature sometimes manages it spontaneously by shutting out one image by a spasmodic ptosis, a method which we have imitated for years by our exclusion pads and opaque lenses. Noyes used to relate a case where he enucleated one eye, on the patient's insistance, for an intolerable, asthenopia, and I have heard the formation of a traumatic cataract suggested as less radical and equally effective. Patients always object to exclusion pads for cosmetic reasons. They may be extremely useful from the diagnostic point of view, however, since symptoms relieved by the exclusion of one eye are almost certainly due to binocular incoördination. The use of the pad also accustoms the patient to depending on one eye and in time makes the

fusion impulse so weak that the pad need be no longer worn. One of the simplest ways of weakening the fusion impulse short of complete exclusion of one eye is by making one retinal image much less distinct or quite different from the other. Keeping one eye under atropine or the wearing of a colored glass has this effect in suitable cases. Dr. Snell has recently suggested what he calls a "meshed" glass one with a series of lines etched on one surface and so graduated as to reduce vision accurately to any degree desired, which ought to be most useful as it does exactly what we have in mind without cosmetic ill effect. Exactly the same result can be secured by using convex spherical or cylindrical lenses strong enough to blur vision. An over correction of two dioptres will reduce distant vision approximately from normal to $\frac{20}{200}$.

This effect can be increased by roughing the back surface of the lens with fine emery paper. This has the advantage that one can graduate the opacity by actual experiment at the time and can if desired limit it to certain portions of the lens according to the effect sought.

In presbyopic patients it is often sufficient to teach the patient to wear his distance correction over one eye and his reading over the other—reversing the lenses in a second pair if desired. If the patient has a distinctly dominant eye it is usually better to put the distance correction over this. This plan has been very effective in several cases of paralysis of one externus where the crippled eye is often in the natural reading position which requires a very limited motion, while the reading lens is almost as effective as a pad in excluding the false image when looking away. There have been a number of patients with marked convergence insufficiency who have been able to wear the usual distance correction allowing fusion of distant images while a bifocal over one eye alone makes fusion of near images unnecessary. This can be reversed in a second pair if desired so as to give the ciliary muscles an equal amount of work.

With younger patients I have had very little experience in suppressing fusion, partly because of reluctance to put it into effect in the young so long as there was any hope of other solution and partly because it is very much more difficult.

Of the methods which are directed at the fusion centers

themselves most are likely to be more useful when combined with those above. In general they reverse the methods useful in building up fusion—separating images by prisms till they cannot be combined and training the patient to study one and disregard the other, just as one trains himself to use a microscope or an ophthalmoscope with both eyes open. The amblyoscope and the stereoscope can be made just as useful in producing disassociation of the eye as for the more usual purposes.

In conclusion let me say again that I am not suggesting these procedures for routine use but merely as alternative ways of handling occasional difficult cases. Neither must it be inferred that all patients take kindly to them.

THE SNELLEN SYMBOL AND VISUAL VALUE.

By Dr. J. A. C. GABRIELS, Albany, N. Y.

A PROBLEM admittedly unsettled and urging solution, any approach to a better understanding or an acceptable interpretation of the values attaching to the Snellen principle of test-type construction and notation, is withal a cherishable ideal and one not too lightly to be waived. This in mind, a new conception is herein offered of the value residing in various degrees of visual acuity as denoted by the Snellen symbol.

In the ensuing plan it will be our purpose to follow along definite lines, setting forth appropriate deductions and conclusions, and illustrating, above all, a method whereby it is hoped to establish that the value expressed in the visual symbol possesses a fixed and characteristic constancy, and that it is indeed independent, genetically and otherwise, of any assumed or so-called arbitrary limit.

The ability of the eye to distinguish letters or other geometrical characters occupying a square, each side of which subtends an angle of 5′ of arc— its component being discernible under a minimum angle of 1′ of arc—has been determined by Snellen to represent normal acuity of vision. Upon this basis the Snellen test types are founded.

In conformity with the principle enunciated, it may be at once recognized that in measuring the value of vision, as interpreted by the Snellen test, we are dealing with figures of more than one dimension, and therefore are concerned not with dimensions themselves but rather, in point of fact, with the surface or area corresponding. Now, the area of a surface is defined as the number of units of surface it contains,

and is equal to the product of its two dimensions. The unit of surface is a square, and, according to definition, a square is a figure both equilateral and equiangular, that is, bounded by four equal sides and having four right angles, and its area is equal to the square of one of its sides.

In the light of the foregoing, let us here look into visual ratings properly to be accredited to the Snellen notation. In the case of the norm or standard Sn. $\frac{1}{1}\frac{0}{0}$, for instance, we have an object from the terminal points of any side of which lines carried to the nodal point of the eye subtend an angle of 5′ of arc, or an angle of 1′ of arc for the component. For this standard test-object, the surface or area value is, of course, one or unity. That is, Sn. $\frac{1}{1}\frac{0}{0}$ has a value of vision equal to one or 100 per cent.

What, now, is the value denoted by the symbol $\frac{1}{1}\frac{0}{0}$? Given the Snellen notation $\frac{1}{1}\frac{0}{0}$ (7.5′), there is indicated an increase or addition to the normal visual angle of 2.5′ of arc. In the 7.5′ of arc resulting, the standard angle has been increased to once and a half its size with a reduction in value, inversely, to two-thirds, or a value for the specific angle increase of one-third. Now a square whose linear measurements are equal to one-third, would be one-ninth, or the square of one of its dimensions, in value of the whole surface or area. Or, given a square each side of which subtends an angle of 7.5′ of arc, its surface or area will be equal to the square of any one of its dimensions. Likewise, a square whose dimensions subtend each an angle of 2.5′ of arc, will have a surface value corresponding to the square of one. Since these are to each other as the squares of one of their sides, it will be found that the area or surface of the former is nine times that of the latter, that is, the second is contained within the first nine times or is one-ninth in value. Accordingly, Sn. $\frac{1}{8}\frac{0}{0}$ expresses a corresponding loss in the value or efficiency of vision of one-ninth, or 0.111, *i.e.*, 11.1 per cent.

Similarly, in the Snellen symbol $\frac{1}{1}\frac{0}{0}$ (10′), there is indicated an increase or addition to the norm of 5′ of arc. In the resultant 10′ of arc, the standard angle has been increased to double or twice its size with a consequent decrease in value, inversely, to one-half—a value, then, for the specific increase of angle of one-half. A square any dimension of which is one-half will

have a value of one-fourth, *i.e.*, the square of one of its dimensions, in the surface or area of the whole. In other words, a square whose linear measure subtends an angle of 10′ of arc has a surface or area equal to the square of one of its dimensions. Likewise, a square each of whose sides subtend an angle of 5′ of arc will be equal in surface or area to the square of one. Thus it will be found that, as each is to the other as the square of one of their sides, the area or surface of the first or given whole is four times that of the second, *i.e.*, the latter is one-fourth the value of the former. Hence, Sn. $\frac{10}{40}$ expresses equivalently a loss in the value of vision of one-fourth, *i.e.*, 0.25 or 25 per cent.

Also, in the Snellen symbol $\frac{25}{10}$ (12.5′), the standard angle having been increased to 12.5′ of arc, there is a reduction in value, inversely, to two-fifths, with a value therefore for the increase in angle of three-fifths. A square whose linear measurement is three-fifths will be, in value of the given surface or area, nine-twenty-fifths. That is, with dimensions for the whole subtending 12.5′ of arc, and for the specific square 7.5′ of arc, the latter represents a surface or area value in the given whole of, decimally, 0.36 or 36 per cent. Continuing, the principle of procedure in the determination of the loss or impairment in vision for other such notations is fundamentally as submitted.

It follows, then, that decrease in vision may be said to vary as the square of increase in distance or angle,—in other words, decrease in value of vision proceeds in the same ratio or proportion with increase in value of surface or area. Thus, it appears that the unit of surface is the true measure of vision. In this connection it should be borne in mind that a surface is, in fact, a collection of points, from each of which rays are directed to the eye and are perceived as distributed in the image formed, and hence visualization of a surface necessarily implies perception of its component points.

Having outlined the method for measuring subnormal values in vision, let us turn to examination of the values indicated by such symbols as $\frac{10}{8}$ and $\frac{10}{18}$. In the notation $\frac{10}{15}$, for example, there is designated a reduction in the standard angle to 3.75′ of arc, a decrease to three-fourths, with a resulting increase in value, inversely, to four-thirds, or a value for the

specific decrease in angle of one-third. A square, therefore, whose dimensions are one-third in value, has a corresponding surface or area value in the whole of one-ninth. Or, a square whose dimensions subtend each an angle of 3.75′ of arc will be to a square the sides of which subtend angles of 1.25′ of arc, as nine is to one, *i.e.*, the surface or area of the latter is one-ninth that of the former. Sn. $\frac{3}{1\frac{8}{8}}$ represents, then, an actual increase in vision of one-ninth or 11.1 per cent., or a visual acuity value of one and one-ninth, *i.e.*, 1.111 or 111.1 per cent.

In the same manner, Sn. $\frac{1}{1\frac{8}{8}}$ signifies a diminution in the standard angle to 2.5′ of arc, *i.e.*, one-half with a consequent increase in value, inversely, to two, or a value for the specific decrease in the given angle of one. The surface or area corresponding to the specific decrease is, with respect to the given surface or area, a value of one. In short, a square with linear measurements subtending 2.5′ of arc will have a surface or area equal to another square of corresponding dimensions, *i.e.*, each is to the other as one is to one. Sn. $\frac{1}{1\frac{8}{8}}$ indicates an actual gain in the value of vision of one or 100 per cent., or a visual value of two or 200 per cent.

In connection with consideration of visual evaluation, the question of the Snellen formula as a fractional unit naturally arises. Taking the symbol $\frac{1}{1\frac{8}{8}}$ (5′), normal acuity of vision may be described as the ability to read 20 foot letters from a distance of 20 feet. The symbol $\frac{1}{1\frac{8}{8}}$ (20: 20) indicates, therefore, a relation or ratio of two numerical values, viz., 20 to 20 feet. Sn. $\frac{1}{1\frac{8}{8}}$, on the other hand, by the same token expresses a comparison or relation of 20 to 30 feet; Sn $\frac{1}{1\frac{8}{8}}$, 20 to 40 feet; Sn. $\frac{1}{1\frac{8}{8}}$, 20 to 50 feet; etc. In the case of Sn. $\frac{1}{1\frac{8}{8}}$, the relation or ratio is of one to one, or unity. In the symbol $\frac{1}{1\frac{8}{8}}$ the ratio is two to three, or two-thirds; in $\frac{1}{1\frac{8}{8}}$, it is two to four, or one-half; in $\frac{1}{1\frac{8}{8}}$, two to five or two-fifths, etc. Such Snellen symbols are inherently fractional values, and reveal, as they do a progressive reduction in value of the standard. In the capacity of true fractions, it may be queried whether such formulæ are capable of subdividing the unit of standard value. It is obvious that $\frac{1}{1\frac{8}{8}}$, $\frac{1}{1\frac{8}{8}}$, $\frac{1}{1\frac{8}{8}}$, etc. cannot be aliquot or equal parts of unity represented by $\frac{1}{1\frac{8}{8}}$, as such. In the units $\frac{1}{1\frac{8}{8}}$, $\frac{1}{1\frac{8}{8}}$, $\frac{1}{1\frac{8}{8}}$, etc., however, standard value is equally connoted,

the subtending angle remaining unaltered just as for the unit $\frac{20}{20}$. Thus, Sn. $\frac{30}{30}$ has a corresponding unity of $\frac{30}{30}$, Sn. $\frac{40}{40}$ of $\frac{40}{40}$, Sn. $\frac{50}{50}$ of $\frac{50}{50}$, etc., each being, in matter of fact, simply an

TABLE.

Visual Symbol of Snellen.	*Visual Loss in Per Cent.*	*Visual Efficiency in Per Cent.*
20/20	0.0	100.0
20/25	4.0	96.0
20/30	11.1	88.9
20/35	18.4	81.6
20/40	25.0	75.0
20/45	30.9	69.1
20/50	36.0	64.0
20/60	44.4	55.6
20/70	51.0	49.0
20/80	56.3	43.7
20/90	60.5	39.5
20/100	64.0	36.0
20/120	69.4	30.6
20/140	73.5	26.5
20/160	76.6	23.4
20/180	79.0	21.0
20/200	81.0	19.0
20/220	82.6	17.4
20/240	84.0	16.0
20/260	85.2	14.8
20/280	86.2	13.8
20/300	87.1	12.9
20/320	87.9	12.1
20/340	88.6	11.4
20/360	89.2	10.8
20/380	89.8	10.2
20/400	90.3	9.7
20/500	92.2	7.8
20/600	93.4	6.6
20/700	94.4	5.6
20/800	95.1	4.9
20/900	95.6	4.4
20/1000	96.0	4.0
20/1100	96.4	3.6
20/1200 (5°)	96.7	3.3
360 degrees	100.0	0.0

expression denoting one or more equal parts of a linear unit. As to symbols such as Sn. $\frac{20}{15}$ and Sn. $\frac{20}{10}$, reference is duly and conformably to unity manifested in $\frac{15}{15}$ and $\frac{10}{10}$, respectively.

In conclusion, the rationale outlined establishes a uniform

and exact method for the evaluation of vision as expressed in Snellen formulæ, while at the same time rendering practicable and universally applicable a record, in decimal or percentage equivalent, of the actual value corresponding to any visual symbol. Study of values so elicited, in orderly tabulation, discloses at a glance wherein lies the vital range of vision, and demonstrates how the curve of visual valuation evolves, bearing evidence meantime of the striking parallelism between acuity of vision, for instance, and the anatomic structure of the retinal perceiving layer.

Subjoined is a specimen scale of Snellen symbols, showing corresponding visual loss and efficiency evaluation in per cent.

SUMMARY.

The unit of surface and not the visual angle is the true criterion in the determination of the value of vision. That is, the actual or absolute measure of vision is based on evaluation of the units of surface rather than of the corresponding linear units.

The value of vision decreases in corresponding proportion with the increase in value of surface or area, and, conversely, increases in the same ratio or proportion with the decrease in value of surface or area.

The Snellen notation treats, in last analysis, of linear units, and hence is not per se the index of visual value.

The Snellen visual symbol is a true fraction.

SOME NOTES ON THE PROBLEMS AND RESULTS OF THE JEWISH CAMPAIGN AGAINST TRACHOMA IN PALESTINE.[1]

By Dr. ARIEH FEIGENBAUM, Jerusalem.

(With two figures in the text.)

A CAMPAIGN against infectious eye diseases in general and trachoma in particular must be based on precise knowledge of the general and local conditions of their origin. Epidemiologically trachoma may be classed as a folks' disease, owing to localization of prevalence, a poor man's disease, the indigent forming the majority of sufferers, a children's disease, as the infection occurs—at least in these regions—in early childhood; and lastly, for obvious reasons, a family disease.

Investigations conducted independently by Auerbach and myself determined that in Palestine two-thirds—three-fourths of the cases of trachoma amongst adults are due to infection in the first three years of life, and the remaining one-fourth—one-third in the following few years. Relatives, particularly the mother suffering from eye-disease, are the main source of infection to the child.

The infectiousness of trachoma is generally exaggerated. Certainly occasionally cases occur of accidental infection; clinical observation indicates, however, that a number of unfavorable circumstances must be present simultaneously in order to provoke infection. These conditions exist plentifully amongst the poor, where florid trachoma is mostly

[1] Paper read 2.14.26 at the First Joint Conference of the School Physicians of the Hadassah Medical Organization and the Teachers Organization. (Abbreviated for publication).

found. Evil housing conditions, common bedclothes, if any, common linen, children uncared for, creeping on the floor, wandering from arm to arm, all contribute to infection.

The following figures relate to three groups of schools attended by children of different social milieu and show that the prevalence of trachoma varies in inverse proportion to the standard of living.

ACTIVE TRACHOMA IN JERUSALEM SCHOOLS (last examination).

Talmud Thorahs	Elementary Schools	Secondary Schools
13%	6.1%	0.9%

In the Hebrew Gymnasium at Tel-Aviv, out of 863 pupils, no cases of active trachoma occur, in the Haifa Realschule (1925) only one case occurred amongst 250 pupils. It is reported from Egypt where trachoma is exceptionally widespread (over 95%) that during 20 years no European oculist has been infected and that the British army was practically free of the disease.

Trachoma, resulting from a low standard of civilization, may almost be called a disgrace if we be permitted to call poverty, ignorance and indolence a disgrace.

Occurring simultaneously with other infectious eye-diseases already dangerous in themselves (mixed infection) trachoma is particularly destructive. Thus from my observation of 3,389 cases of acute ophthalmia in 1916 I observed that trachoma cases affected by the otherwise comparatively benignant Koch-Weeks ophthalmia tend more to complications of the cornea (13.1%), often to especially serious forms, than non-trachomatous (3.1%). The adverse influence of summer conjunctivitis on trachoma will be made apparent later in this paper. We may regard as *one enemy* demanding a common plan of campaign summer ophthalmias caused by B. Koch-Weeks, autumnal ophthalmias caused by the Gonococcus (which I observed 156 times amongst 3,561 smears during a period of 3½ years) and the other infectious eye-diseases occurring together with trachoma. A trachoma campaign therefore, must be directed against all infectious eye-diseases.

The rational plan of action against trachoma obviously

includes increased prosperity of the country, raising the general standards of sanitation, improved means of communication, increase of the number of schools and so on. Viewing the problem purely from the medical side, however, we do not arrive at the root of the evil. Early treatment, continued energetically until complete cure, can as we shall see later, bring excellent results; on the other hand, we must confess that, by treatment only, we try to fill a bottomless cask; after treatment is stopped for a while the disease reappears unabated. The problem then is not purely therapeutic but also and principally one of social hygiene. All methods of attack must be undertaken simultaneously. None are so unimportant as to be disregarded. This applies at least, may be, unless a specific cure, sure and rapid, is discovered. In this connection we may possibly await help from Radio-Therapy.

As to necessities,—a good water supply is of first importance and in this regard Palestine generally, and Jerusalem particularly, suffers. Under the present administration a favorable solution of this aspect of the problem may be expected.

Propaganda on the need for sustained treatment of the disease is required. Oral instruction in the elements of preventative hygiene is more valuable than written, which, where unavoidable, requires the simplest and clearest form of expression. Superstitions, very prevalent, need to be obstinately fought. Our endeavors in this direction depend directly on the support of the teacher and the trained District Nurse.

Further, medically, development is essential in a systematic and centrally controlled scheme of action. An adequate cadre of oculists is required as well as working knowledge of the prevalent eye-diseases on the part of the general practitioner; a sufficient number of eye hospitals and clinics and training of nursing personnel are necessary and courses on diagnosis and treatment of trachoma must be instituted. Great progress has been made during the last 12 to 13 years in this respect. This may be ascribed, in particular to the work done by the Hadassah Medical Organization, the well-known American Jewish Institution. For the first time eye-departments were founded in Jaffa, Haifa, Tiberias and Safad, and a good school for nursing was established in connection with

the Rothschild-Hospital (Had. Med. Org.) in Jerusalem. Systematic school treatment has been instituted all over the country. The number of oculists has increased.

In spite of the still unsatisfactory situation a great deal has been carried out of a comprehensive programme which I drew up in 1913 for combating trachoma, part of which is recapitulated herein. Confidence in the treatment increases amongst the habitants. Whereas previously the treatment, although offered free of charge, was scarcely tolerated, it is now generally being recognized even by the simplest folk as a necessity.

It is very necessary to make treatment as easy as possible for the patient, demanding a minimum of his energy. For this purpose the establishment of free district-clinics in districts where the disease is rife, is recommended. In Jerusalem, in 1913, I instituted two such clinics and subsequently a third, and supervised their working up to 1919 when they ceased to exist. It is hoped that they will be reopened in the near future by the H. M. O. In rural districts such clinics are required in each settlement, the work being subordinated to the central body and control adumbrated above. A step towards this desideratum had been taken in as far as the Jewish population is concerned by means of a travelling oculist of the Hadassah Medical Organization, who supervises the systematic work and endeavors to create and subsequently to sustain the active interest of the local practitioner in the trachoma campaign.

From the therapeutic and from the pedagogic standpoint the eye treatment of the Hadassah Medical Organization in Jewish Schools as well as by the Government in recent years, in Arab schools, deserves special mention. Excellent results have been obtained and the treatment has become an important branch of social hygiene. In the school which can, however, scarcely be considered as spreading-places of trachoma, we find a solid group of patients who can be treated daily over a prolonged period until cured. In spite of the therapeutic success, it must be remembered that we come too late with true *prophylaxis*, since the majority of cases are infected before Kindergarten age. The institutions undertaking general hygiene and prophylaxis work amongst in-

fants, organized and conducted by the Hadassah are, therefore, of particular importance. In conjunction with these institutions the rational eye-prophylaxis including prenatal care should find its natural place. By this means contact is established with families where trachoma is prevalent. This supplements the tracing of trachoma cases to families through school-children. It is hoped that the necessary steps will be undertaken by the H. M. O. in the near future.

Our school-treatment is arranged as follows: The school oculist examines all children, at least twice yearly, at the beginning of the term and records in case of disease the stage and treatment prescribed. These records are simplified by using abbreviations for the different stages and differentiating active from cicatriced trachoma. The treatment is carried out daily by trained nurses and is continued until the next examination of these cases, which takes place every 4 to 6 weeks. Any doubtful cases occurring during this interval are sent to the clinic and returned to the nurse with a revised prescription. The appropriate clinics undertake at the same time all surgical intervention. Every endeavor is made to continue treatment during the holidays.

I append some figures relating to Jerusalem schools under my supervision:

Year	Examined	Active trachoma	%
1913–14	5936	1461	24.6
1921	5433	918	16.9
1925	5011	579	11.5

The above figures represent mere steps of the steadily continued decrease in active trachoma. It will be realized that the number of children passing through the schools during this period is very considerable and that the diminution of 13.1% represents far greater progress than at first appears.

In Jerusalem immigration does not, it should be remembered, reduce the number of trachomatous children; on the contrary, the immigrant children (from Persia, Kurdistan, Mesopotamia, etc.) add considerably to the figures; *e.g.*, in the beginning of 1925 of 5011 pupils examined 11.5% were suffering from active trachoma and 10.8% showed signs of past

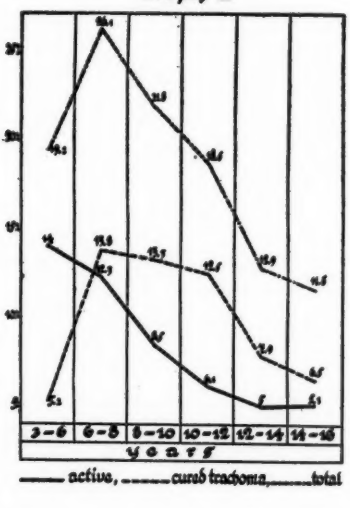
trachoma. This figure includes 921 new pupils of whom 13.2% had active and only 7.1% cicatriced trachoma.

The progress in the other stations in Palestine is set out below in the following table, referring to the Jewish population and active trachoma showing the position in 1913–14 (excepting Jaffa and Safad 1921) and 1925.

	Trachoma in %	
	1913–14	1915
Jaffa (1921)	34	12.1
Haifa	23	8.5
Tiberias	83	44.5
Safad (1921)	26.5	27
37 Jewish settlements (average)	24.4	11.3

The regular treatment produces a twofold result: a considerable number of sources of infection are closed and by

Graph I



dealing with young individuals we avoid the evil results of trachoma, not treated early and energetically enough. Since

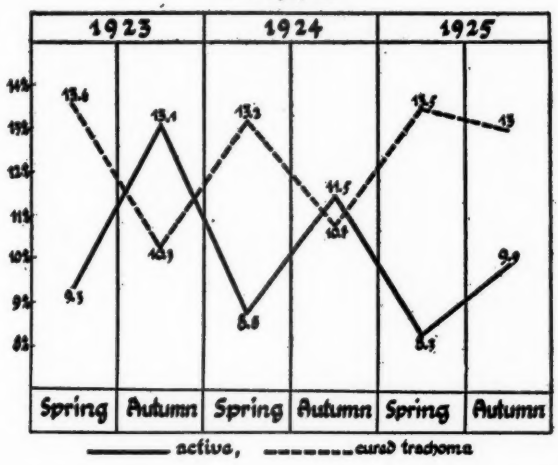
the systematic treatment of school-children has been undertaken far fewer serious cases than previously are encountered.

The results of the treatment can be demonstrated yet otherwise. The following graph shows the division between cured and active trachoma amongst 5,360 school-children, arranged according to age (summer 1923 examination); it exemplifies how so complete a cure may be effected by systematic treatment as not to leave a trace of infection (cicatrices and pannus), a fact, to which too little attention is generally paid.

After a natural rise in the beginning, a diminution of active trachoma occurs as age increases, not followed, however, by a corresponding increase of cicatriced cases.

Our examination in Jerusalem schools also shows very clearly the aggravating influence on trachoma of the annually

Graph II



recurring summer conjunctivitis. After the epidemic has run its course, cases of trachoma which were almost cured, again become active, consequently increasing the total of active trachoma cases. The following graph containing results of examinations (spring and autumn) over a period of some years illustrates this fact:—

In addition to summer ophthalmias the school holidays tend to increase active trachoma in the autumn. During this period naturally treatment cannot be continued to the same extent. Trachoma, a most obstinate disease, can only be combated by like obstinacy. Frequently a slight interruption in treatment causes relapse to a stage from which we had advanced after months of sustained effort.

Finally I have observed that diseases of the nose exercise an aggravating effect on trachoma and add yet another factor for consideration in the general plan of campaign against the disease.

METHOD OF THREAD FIXATION OF THE LID IN TARSUS OPERATIONS.[1]

By Prof. Dr. L. de LIEBERMANN, Budapest, Hungary.

Edited by Dr. W. J. Werfelman, Jr., Syracuse, N. Y.

(With one illustration in the text.)

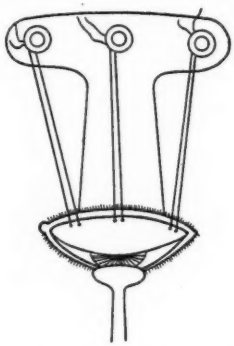
WHEN Prof. Blaskovics first demonstrated his method of turning the tarsus in the Hungarian Ophthalmological Society, long before he published the detailed description of this operation, I adopted it as the best or rather, as the only good operation for cicatricial entropion caused by trachoma. At the time when his German paper appeared, I had gained enough experience with the method to give a report on it shortly after, and to be able to recommend it highly, and also to offer a method to make the technique easier and safer, a modification which the author himself accepted and quoted as an improvement, namely, the use of threads for fixing the lids. My publication of it appeared and after discussing it personally with Prof. Blaskovics, I found that he was agreeable to my method. I considered it necessary knowing that without this help the operation was a rather difficult one, and being such, we could not expect it to gain the popularity that it would deserve. Certain faults that almost every surgeon would discover when first trying it, would carry the danger of spoiling their favorable opinion of this method. I find that through all the years that have passed since Prof. Blaskovics published his operation, no modifications to his method have appeared in the *World's Ophthalmic Literature.* I have observed that the new German text book of *Ophthalmic*

[1] With references and remarks concerning Dr. Blaskovics' paper on his turning of the tarsus operation.

Surgery, and works by other German authors, have asked for suggestions or modifications to this operation. I feel that my paper is the only one on this subject, unless something appears before mine gets into the literature.

Yet the difficulties of this operation are not so great that the surgeon would be justified in ignoring the method, which is, I wish to emphasize again, the only good operation for this particular condition. I said this previously when I had to speak about it in my paper on my cautery excision method, a paper to which I will now refer for another reason. When I first recommended the use of fixing threads in connection with my screw-ended lid plate, and threaded the needles once through the lid margin exactly 1*mm* above the lid margin, and either from the conjunctival surface to the skin surface, or vice versa. The purpose of it was not only to fix the lid upward in its everted position, but to stretch the entropionized lid margin, flattening the curved sulcus subtarsalis, so as to make the most important step of the operation easier.

It proved suitable for the purpose, but when using it oftener, not only for Blaskovics' turning operation, but also for my cautery excision, and the combination of the two oper-



ations, which proved very suitable for most of the cases of cicatricial entropion, I soon noticed that the thread fixing method could be improved simply by leading the needle from the conjunctival surface to the outer (skin surface),

and *2mm* from it, back again so that needle and thread emerged on the conjunctival surface of the lid margin, the loop thus lying on the skin. When pulled upward and fixed on the three screws at the T-shaped end of my new lid-plate, the three double threads stretched in divergent directions are stretching the whole tarsus and especially its entropionized border completely flat, still more than those applied in the first way. I explained it in the paper dealing with my cautery excision method. Some two years later an assistant of Prof. Blaskovics, giving a report of the results of the turning of tarsus operations performed in Prof. Blaskovics' Hospital, speaking of technique said that after using the fixation threads they observed sometimes a small partial necrosis of the lid margin, and therefore Prof. Blaskovics tried to avoid threading through the tarsus margin, and placed the fixation threads through a small skin fold only.

My answer to this remark was given in a paper published in 1924, in which I pointed out what briefly follows: They have forgotten what I recommended in the above paper, namely, the stitching back with the needle to the conjunctival surface. No lid margin necrosis occurred in any of my cases when I used the first method, however, it may be possible, the lid margin vessels being strangulated by the threads if strongly pulled. But using my new method this is quite impossible, the emerging of both thread ends at the conjunctival surface does not only avoid this danger, but follows Blaskovics' method by facilitating the exact placing of the tarsus cut. The threads can do no harm to the lid margin vessels, as in this way they are not lying in a strangulated loop. At least the threads never caused the least harm in my hands. Fine needles and fine thread are to be used.

I was surprised to see that after all these explanations and discussions in several papers, Prof. Blaskovics in his latest paper said the same thing about the thread fixing methods. He himself reported a number of mistakes made by not succeeding in cutting the tarsus at the right angle. He discarded the safest way to avoid this difficulty, instead of applying this in the right way. Perhaps one can succeed as well with his modified big blephorostat, though I should not think it as easy, the author emphasizing it again on page 42, that

the tarsus being bent in an angle—the blade of the knife will form during the section a pointed angle with the convex edge of the tarsus. Avoiding this difficulty is the chief advantage of the thread-fixing method, beside others, and therefore I cannot see any reason to drop a method that proved satisfactory, not only in the turning of the tarsus operation, but in almost every lid operation, on either upper or lower lid, either on the skin or conjunctival side, which is absolutely harmless if done in the proper way.

I have to lay some stress on these facts as I do not want to have the thread-fixing method considered only as a help to the Blaskovics operation, but as a method which can be used to advantage in almost any lid operation.

BIBLIOGRAPHY.

Arch. of Ophthalmology, vol. 54, No. 1, p. 38.
Zeitschr. f. Augenheilk, vol. 28, p. 286, 1912.
Klin. Monatsbl. f. Aug., vol. 61, p. 577, 1918.
Klin. Monatsbl. f. Aug., vol. 61, p. 660, 1918.
Graefe's Arch. f. Ophth., 105, p. 542.
Klin. Monatsbl. f. Aug., vol. 72, p. 209.
Arch. of Ophth., vol. 54, p. 38.

THE SURGICAL TREATMENT OF WOUNDS OF THE CORNEA WITH PROLAPSED IRIS.[1]

By Dr. P. CHALMERS JAMESON, Brooklyn.

(With five illustrations on Text-Plates XXIV. and XXV.)

THE object of this communication is not to review or cover the entire subject of the treatment of wounds of the anterior portion of the globe (this has been excellently dealt with in recent papers, notably those of Mr. Charles B. Goulden of London and Mr. J. C. Clegg of Manchester) but to present to you two surgical procedures which the writer has found valuable in his experience in the treatment of wounds of the cornea with and without prolapsed iris.

Clean cut wounds of the cornea frequently present themselves for treatment and in these the prognosis is best. It almost goes without saying the wound should be carefully and microscopically inspected and the nature of the implement which made the wound ascertained. Debris carried with the wound should be removed and the parts well irrigated with a mild solution of saline or boric acid. It is well to avoid strong cauterizants such as carbolic acid and iodine unless their use, by reason of infective instruments, is necessarily suggested, but nitrate of silver, ½ of 1% can be followed by saline douche. The supportive bandage is nearly always helpful, acting as a splint and promoting coaptation of lip wounds. The gaping of the wound is not always apparent immediately after the injury, but may evidence itself more, later, by the giving way of new formed tissue in its effort to hold back the restored anterior chamber;

[1] Read before Am. Ophth. Soc., Hot Springs, Va., June, 1926.

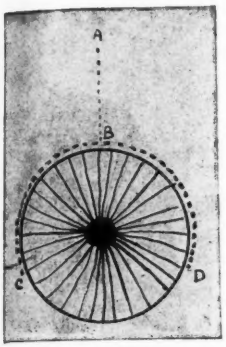
thus the conjunctival flap is of great utility, and in large wounds of the cornea it is nearly always indicated.

The requisite of a good conjunctival flap is that it should make even pressure throughout an area well extended in every direction beyond the line of the wound incision, that it should not be a taut string or strip likely to indent the line of the wound thus defeating its object; that it should not slip; that it should leave the conjunctival tissue intact without unnecessary excision; and also that it be a flap with which the suture material will not come in contact nor shall it irritate the cornea.

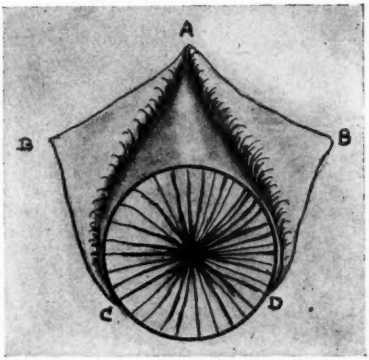
The surgeon will select the flap which is best adapted to his purpose, but I take this opportunity of presenting a procedure of conjunctival flap which has a wide range of utility and is adapted to almost any emergency in which a flap is necessary. It fulfills all the above requisites mentioned and can be adapted to cover a small portion or to cover the entire surface of the cornea if desired.

I designate it as the double triangular flap. The method is as follows. Three primary linear incisions in the conjunctiva are necessary. One straight incision starting from a point at the limbus corresponding to the top of the vertical meridian of the cornea is carried back directly to the fornix and two curved incisions around the limbus, one on the inner and the other on the outer side. The extent of these curved incisions depends on the size the flap required, which of course rests upon how much cornea, is to be covered. Variation as to the point on the limbus from which these incisions start can be made; but, beginning as above stated, a flap can be obtained to cover the entire cornea. The triangular flaps are now undermined from the points of the vertical incision at the limbus which form the apices of the triangle to their bases. This gives us two distinct flaps and a plentiful supply of conjunctiva. The apex of the first flap is drawn over the cornea and sutured to the sclera at a point corresponding to the lower terminus of the limbal incisions on the opposite side from which the flap is derived and it is sutured to the episclera by two or three sutures. This firmly secures the base flap in position covering the cornea. The apex of the other flap, the superficial or supporting flap, is drawn over the cornea in

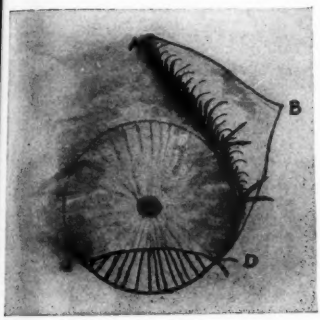
ILLUSTRATING DR. P. CHALMERS JAMESON'S ARTICLE, "THE SURGICAL TREATMENT OF WOUNDS OF THE CORNEA WITH PROLAPSED IRIS."



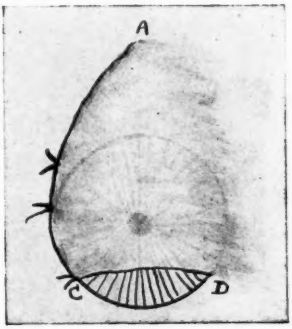
1. Incisions, A B, fornix to limbus.



2. Triangular flaps A B C and A B D turned back.



3. Base flap sutured, apex B of the base flap A B C sutured to lower end of limbal incision D.



4. Complete triangular flap. Apex B of superficial flap A B D sutured to lower end of limbal incision C.

exactly the same way, to the opposite lower terminus of the limbal incision and sutured to the conjunctiva at the limbus, or conjunctiva and episclera. The edges of both of these flaps corresponding to the limbus incision are free which admits of drainage through the wound underneath. The two triangular flaps thus sutured in situs can be described and outlined by drawing two lines from the top of the incision at the fornix to the lower end of each limbal incision: the bases by a line drawn from the ends of each limbal incision. The two together—base and superficial flaps support and cover the cornea with a double layer of conjunctiva. After having fulfilled their functions of support, they dispose of themselves in the following manner.

The superficial flap pulls out in some three days and retracts to its original anatomical position and melts out of sight leaving no disturbance or ruffling of the area from whence it was derived. The base flap sutured to the sclera holds longer but in turn pulls out, retracts to its proper structural position, reunites leaving in each instance little disturbance or ruffling. There is then no excision of conjunctiva and no tissue lost.

The second procedure which it is the object of this paper to commend is utilized in the treatment of prolapsed iris following corneal wounds.

The surgical treatment, of course, depends somewhat on the location of the wound and prolapse, upon the time that has elapsed from its reception and upon the extent of the trauma and maceration.

A prolapsed iris, if seen early, the neck of which is not firmly constricted or agglutinated, can sometimes be replaced by gentle pressure with a spatula from without, and with the help of eserine or atropin posterior incarceration may be avoided. This certainly should be attempted before excision.

Constriction at the neck of a balloon prolapsis, with agglutination and strangulation, however, occur very rapidly within a few hours. The inclination is to excise the prolapsis and this is undoubtedly the popular method. It, however, has some disadvantages. Excision leaves an unsightly coloboma and also opens the vessel system of the iris.

The method of replacement of prolapsed iris by traction

from within the anterior chamber, used in selected cases, is attended with most gratifying results and may restore the iris to its normal position intact. The principle of this method I will present later.

In 1909 in the ARCHIVES OF OPHTHALMOLOGY I wrote a short report on the advantages of replacement of the iris by traction on the neck of the prolapsis from within the anterior chamber. After publishing it I found that Dr. John Dunn has previously reported an operation by which replacement was accomplished by a large peripheral corneal section and traction with a spatula three days after the prolapsis had taken place, and in recent years Mr. C. B. Goulden of London used a like method.

The procedure I advocated is as follows:

A counter-incision far enough away from the point of injury so as not to interfere with nutrition is made in the cornea. A delicate, malleable blunt-pointed probe, bent to any curve or angle desired, can be made to engage the neck of the prolapsis on the posterior side through this opening. Gentle pressure from without with a spatula and slight traction from within the anterior chamber, will often reduce a prolapse, even, if three to five days have elapsed.

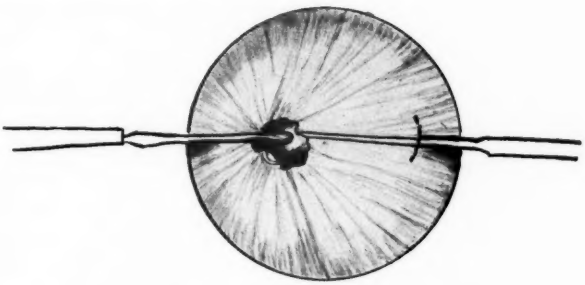
The advantage of this method of reduction, when it can be accomplished, are numerous.

1st. It admits of most delicate manipulation substituting two forces for replacement instead of one, viz.: gentle pressure from without the anterior chamber and well regulated traction from within the chamber.

It thereby preserves the iris from undue maceration and protects the corneal lips of the wound from injurious traumatism.

2nd. The counter-incision in the cornea, does not alone serve the purpose of introduction of the probe for traction but contributes another valuable attribute to restoration and healing. If we subject an elastic surface to posteriorly confined water pressure and incise it the aperture will gap. If, however, a counter-incision is made, the lips of the primary incision will more nearly coapt. Thus a counter-incision in the cornea

ILLUSTRATING DR. P. CHALMERS JAMESON'S ARTICLE,
"THE SURGICAL TREATMENT OF WOUNDS OF THE
CORNEA WITH PROLAPSED IRIS."



Replacement of Prolapsed Iris by Traction from within the Anterior Chamber.

after replacement diverts the flow from the primary corneal wound and equalization of aqueous pressure upon the cornea permits the lips of the wound to close.

3rd. The divertion of the flow of aqueous from the single direction of the wound, lessens the danger of secondary prolapsis.

4th. The counter-incision admits of complete and thorough irrigation of the anterior chamber.

5th. If replacement is successful it leaves an unmutilated iris, often a circular pupil, and the cosmetic result is excellent. Incidentally, this principle of counter-opening can be utilized in obstinate fistulæ of the cornea in conjunction with the double triangular flap, as an accessory operative means, to reduce tension while the coapted edges of the curetted fistulæ are healing.

I have never found counter-incision in these cases of prolapsis to do harm and certainly the danger is less than that of opening the vessel system, as in excision and the sometimes severe maceration of iris tissue and wounding of lips in efforts solely from without.

Before replacement the prolapsed iris can be sterilized with ½ of 1% nitrate of silver followed by irrigation of normal salt solution.

The cases best adapted to this procedure of counter-incision and traction within the chamber are the smaller wounds with balloon prolapsis and, of course, the earlier the case is seen, the more facile is the reduction. Cases that present much maceration and laceration of the iris with stellate ragged tear of corneal tissue are not those that lend themselves to replacement by this procedure.

One can with much certainty commend the use of the double triangular conjunctival flap, and in selected cases the replacement of prolapsed iris by counter-incision and by traction from within the anterior chambers.

STUDIES OF THE VISUAL FIELDS IN LOCALIZED CHORIORETINITIS OF THE JENSEN TYPE.[1]

By Dr. HENRY P. WAGENER,
Section on Ophthalmology, Mayo Clinic

And Dr. JOHN F. GIPNER,
Fellow in Ophthalmology,
The Mayo Foundation, Rochester, Minnesota.

(With eighteen illustrations on Text-Plates XXVI.–XXVIII.)

THE characteristic feature of "retinochoroiditis juxtapapillaris" emphasized by Jensen in his original description was the sector-shaped defect in the field of vision. In a paper presented at the Oxford Ophthalmological Congress (1924), Friedenwald gave an excellent summary of the published studies on Jensen's disease and of his own views on the subject. He showed quite conclusively that, from the standpoints of etiology, pathology, and clinical course, all forms of localized chorioretinitis can be grouped together, and that the difference in the configuration of the visual fields in individual cases depends only on the location of the lesion and the degree of involvement of the various layers of the retina. In cases in which there is a sector-shaped field defect, it seems most probable, as agreed by most authors, that the layer of nerve fibers at the site of the lesion has been injured. However, the hypothesis that disease of the retinal arteries secondary to the retinitis results in poor nutrition of the peripheral portions of the retina and thus causes the field defect, still receives some support, even from Friedenwald. In most of the reports of cases of chorioretinitis juxtapapillaris, the disease was active at the time the field was examined. In the

[1] Submitted for publication March 9, 1926.

ILLUSTRATING ARTICLE OF DR. HENRY P. WAGENER AND DR. JOHN F. GIPNER, "STUDIES OF THE VISUAL FIELDS IN LOCALIZED CHORIORETINITIS OF THE JENSEN TYPE."

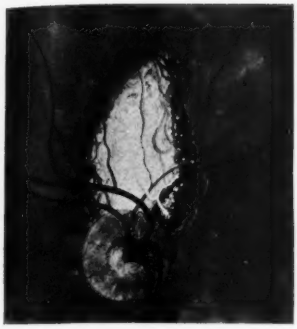


FIG. 1

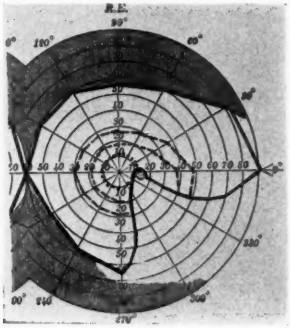


FIG. 2

FIG. 3

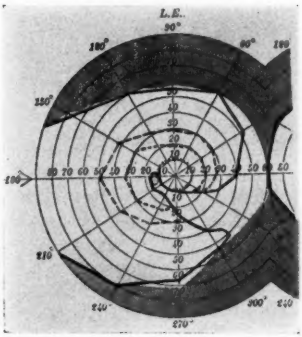


FIG. 4

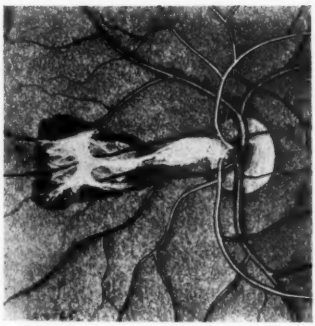


FIG. 5

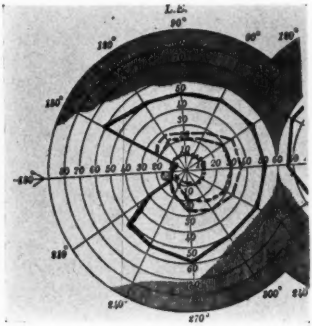


FIG. 6

acute stage, the edema surrounding the actual focus of inflammation alters the field defect somewhat and masks the site of injury to the nerve fibers. After the lesion has healed, however, a comparative study of the retinal scar, and of the resultant field defect should enable one to judge whether a vascular lesion can account for the field defect and also, as van der Hoeve suggests, to estimate, as an adjunct to other clinical and anatomic studies, the course of the nerve fibers in the retina as they radiate from the optic papilla. With these purposes in view the drawings of the lesions of the fundus and the corresponding perimetric fields in six cases of inactive localized chorioretinitis are presented. A case of acute chorioretinitis juxtapapillaris is included in the series, but will not be considered authoritative in the comparative study because of the difficulty of determining the extent of the actual focus.

REPORT OF CASES.

CASE 1.—A married woman, aged thirty-eight, came to the clinic June 27, 1925, because of nervousness, and was referred to the Section on Ophthalmology. At the age of nineteen she had had blurred vision in the right eye caused by an acute choroiditis. She had also, at this time, acute rhinitis and probably sinusitis, which received no special treatment. The vision cleared in about a month, and so far as the patient knew, there had been no recurrence.

Examination.—With the proper refractive correction, the vision in each eye was $\frac{6}{6}$. Externally the eyes were normal. In the left eye the media were clear and the fundus normal. In the right eye there were some fine floating vitreous opacities and, just at the upper margin of the optic disk, a patch of inactive chorioretinitis about 2 disk diameters in size and roughly pear-shaped in outline (Fig. 1). In the central part of this patch the sclera was exposed, but the atrophy of the choroid was less complete peripherally as there were still visible a number of the larger choroidal vessels. The atrophic area was bordered by a zone of proliferated pigment, and there was some diffuse grayish pigmentation in the choroid above the actual lesion. The large retinal vessels crossing the patch were unaltered, the smaller branches showed mild contraction and perivascular thickening. A Y-shaped band of connective tissue projected forward into the vitreous from the part of the lesion crossed by the superior nasal retinal

vein. The optic papilla was normal and there were no lesions elsewhere in the fundus. Fields of vision on the arc perimeter and on the Lloyd stereocampimeter showed a defect in the right inferior temporal quadrant, which included the normal blind spot (Fig. 2).

In the general physical examination of the patient nothing was found which might have a bearing on the condition of the eye except potential foci of infection. The tonsils were enlarged and moderately infected. There was periapical infection of one tooth. There was mild cervical adenitis. The urine and blood count were essentially normal. The Wassermann reaction of the blood was negative. The patient was subject to attacks simulating petit mal, but there was no evidence of an organic cerebral lesion.

CASE 2.—A married woman, aged fifty-one, came to the clinic July 13, 1925, for the relief of menorrhagia. She was referred to the Section on Ophthalmology because she complained of muscæ volitantes. Her history was of little importance aside from the fact that she had had influenza twice and had suffered from recurrent tonsillitis, for which tonsillectomy had been performed. There had been no evidence of reduced vision at any time.

Her vision was $\frac{6}{6}$ in each eye without correction. The eyes were normal externally. In the right eye the media and fundus were clear. In the left eye were some fine floating vitreous opacities and, just above the disk, lying partially under the superior nasal vessels, was a small patch of inactive chorioretinitis. This patch was about one-half disk diameter in size, the sclera was exposed over most of its area, and its upper nasal margin was bordered by a zone of proliferated pigment. There were no choroidal vessels visible in the atrophic spot. There was superficial punctate depigmentation of the choroid for some distance around the main lesion, and perivasculitis of the branches of the superior nasal vessels over the patch; otherwise the retinal vessels were normal. The optic papilla was normal and there were no lesions elsewhere in the fundus (Fig. 3). Perimetric fields, mapped on the arc perimeter and on the Lloyd stereocampimeter, showed a defect for form and color in the left inferior nasal sector. The defect included the normal blind spot (Fig. 4).

General physical examination of the patient was essentially negative except for the presence of rather marked pyorrhœa alveolaris, a small fibromyoma of the uterus, and lacerations of the cervix and perineum. The Wassermann reaction of the blood was negative.

CASE 3.—A married woman, aged thirty-eight, came to the clinic August 12, 1925, because of headaches. Thirteen years before, she had consulted an ophthalmologist on account of seeing spots before her eyes. At that time a defect was discovered in the temporal field of the left eye; she did not recall whether any other abnormalities were found.

At the time of her examination at the clinic she had no ocular symptoms except the field defect in the left eye. The vision in each eye was $\frac{6}{6}$. The near point was 13*cm* in each eye. The eyes were normal externally. In the right eye the media and fundus were normal; in the left eye the media were clear. In the nasal portion of the choroid and retina, there was an atrophic patch about 1 by 2 disk diameters, extending to within 1 disk diameter of the nasal margin of the disk. In this area several sclerosed large choroidal vessels were exposed but no sclera was visible. The patch was entirely surrounded by a rather wide and dense zone of proliferated pigment. In the overlying retina was an irregularly radiating strand of connective tissue which spread over the retinal vessels in the atrophic area and extended over the intervening normal appearing retina and choroid to attach itself to the nasal portion of the optic papilla in the region where the central artery branches. The papilla itself was normal as were the retinal vessels and the remainder of the fundus (Fig. 5). In the temporal field of the left eye there was a sector-shaped defect for form and color which was continuous with the normal blind spot, as shown both on the arc perimeter and on the Lloyd stereocampimeter (Fig. 6).

General physical examination of the patient did not reveal anything of significance with reference to the eye. The Wassermann reaction of the blood was negative. There was no dental infection. The patient stated that she had had dilatation and curettage because of leukorrhea in 1915, appendectomy in 1921, and tonsillectomy in 1922. It is possible that a focus of infection was removed by one of these procedures.

CASE 4.—A man, aged sixty, came to the clinic August 20, 1925, because of pain in the right lower quadrant of the abdomen. He was referred to the Section on Ophthalmology because he saw spots before his eyes. His left eye had been injured three times; in 1893, it was struck by a firecracker; in 1895, by the limb of a tree, and in 1913, by a piece of kindling wood. Following the last injury, the vision was quite hazy for several days. In 1921, he dis-

covered that, if he looked at an object with his left eye alone, he could not see the upper half of it.

With the proper refractive correction, the vision of the right eye was $\frac{6}{6}$ and that of the left $\frac{6}{36}$. The eyes were normal externally except for slight divergence of the left eye. In the right eye the media were clear and the fundus was normal except for mild retinal arteriosclerosis. In the left eye there was a small opacity at the posterior pole of the lens and some floating vitreous opacities. There was definite pallor of the lower two-thirds of the optic papilla. Extending downward and temporally from the lower margin of the papilla, following the course of the inferior temporal retinal vessels to a point below the fovea, was an area of superficial atrophy of the choroid with superimposed irregularly proliferated and clumped pigment. The retinal arteries, in general, were mildly sclerosed. The artery overlying the affected area was considerably contracted, and both artery and vein in this region showed mild perivascular thickening. There were three spots of proliferated pigment grouped in the macular region (Fig. 7). In the visual field of the left eye, as mapped on the arc perimeter and on the Lloyd stereocampimeter, there was a relative central scotoma for form and green and an absence of the entire upper nasal quadrant and of most of the upper temporal quadrant. This field defect included the normal blind spot (Fig. 8).

General physical examination of the patient did not show anything related to the condition of the eye. The Wassermann reaction of the blood was negative. There was no dental infection. Tags of tonsils remained from a previous tonsillectomy. Six years before his visit to the clinic, the right kidney had been removed because of renal stones. Trauma possibly played a part in the causation of the chorioretinitis in this patient; but the foci of infection in the tonsils and in the kidney must be considered as possible causes also.

CASE 5.—An unmarried woman, aged thirty-three, came to the clinic December 8, 1925, because of menorrhagia and "noises in the head." When she was examined in the Section on Ophthalmology, she complained of seeing spots before her eyes and of loss of a part of the field of vision of the left eye. She had first noticed these symptoms following removal of the appendix ten years previously.

The patient's vision was $\frac{6}{6}$ in each eye. Visual fields mapped on the arc perimeter and on the Lloyd stereocampimeter showed, in the right eye, a small absolute scotoma about 10 degrees in diameter situated about 5 degrees above

ILLUSTRATING ARTICLE OF DR. HENRY P. WAGENER AND DR. JOHN F. GIPNER, "STUDIES OF THE VISUAL FIELDS IN LOCALIZED CHORIORETINITIS OF THE JENSEN TYPE."

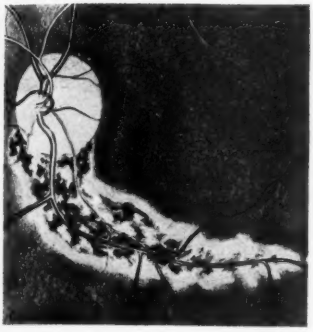


FIG. 7

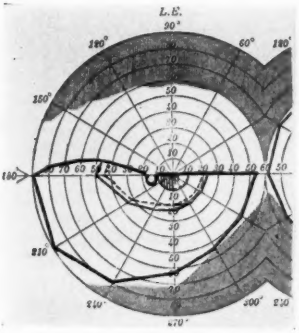


FIG. 8

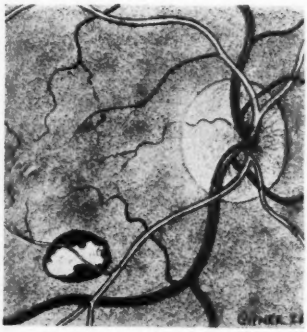


FIG. 9

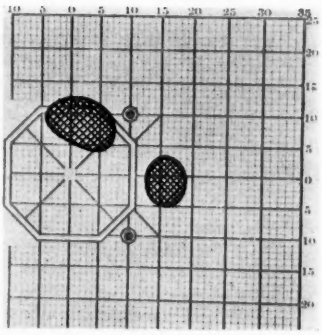


FIG. 10

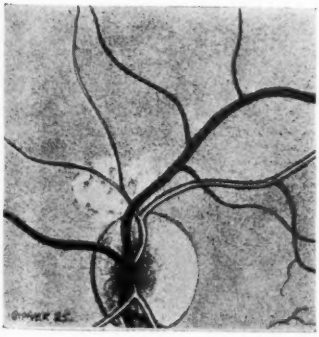


FIG. 11

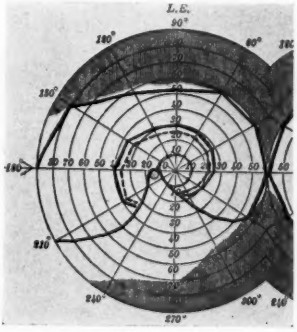


FIG. 12

the point of fixation (Fig. 9), and in the left eye, a lower quadrant defect which included the normal blind spot (Fig. 10). External examination of the eyes was negative. There were a few floating vitreous opacities in each eye. In the right eye, under the inferior temporal vessels about 1 disk diameter from the disk, was a patch of partial atrophy of the choroid of about one-third disk diameter, with a rim of proliferated pigment (Fig. 11). The retinal vessels crossing this path did not appear to be affected. In the left eye, at the upper margin of the disk was a patch of superficial atrophy of the choroid without pigment proliferation, of about 0.5 disk diameter. The superior nasal vessels crossed the patch but showed little evidence of disease (Fig. 12).

General and neurologic examination were negative except for moderate secondary anemia. The erythrocytes numbered 3,080,000 and the hemoglobin percentage was 40 (Dare). The blood platelets numbered 170,000. This low platelet count led to the tentative diagnosis of mild purpura hæmorrhagica. The tonsils had been removed some time previously, but a small tag remained on one side. When this was removed a pus pocket was found behind the posterior pillar. The Wassermann reaction of the blood was negative.

CASE 6.—A married woman, aged twenty-nine, came to the clinic August 24, 1925, because of a gradual change in her facial expression and an increase in the size of her hands and feet.

The patient was found to have adenomatous goiter with hyperthyroidism, mild hypertension, definite acromegaly, and a considerably enlarged sella with erosion of the posterior clinoids. She was referred to the Section on Ophthalmology to determine whether there were associated ocular changes. Her vision was $\frac{6}{6}$ in each eye and her near point was 8*cm.* Externally the eyes were normal. There was a slight reduction of the pupillary reflex to light. The optic disks were small and of good color. There was some excess of glial tissue around the vessels on the disks. In the left eye, under the small branches of the inferior temporal vessels, about 2.5 disk diameters below the disk, there was a scar of an old chorioretinitis of about 1 disk diameter. There was superficial atrophy of the choroid with considerable proliferation of pigment, mainly marginal in distribution. The retinal vessels crossing the patch showed definite perivascular thickening without, however, visible reduction of their lumina (Fig. 13). Fields of vision mapped on the arc perimeter and on the Lloyd stereocampimeter showed in the left eye a defect of the superior nasal

sector extending to within 12 degrees of the point of fixation. The blind spot was normal in size and not connected with the sector defect (Fig. 14).

The patient was not completely examined for possible foci of infection. There was apparently a moderate grade of oral sepsis, but no roentgenograms of the teeth were made. The Wassermann reaction of the blood was negative.

CASE 7.—A married woman, aged fifty-one, came to the clinic November 23, 1925, because of reduced vision in the left eye. In February there had been a severe reaction to vaccination for smallpox and at the same time acute follicular tonsillitis. Two weeks before her visit to the clinic, following a cold in the head, she noticed a blurring of the vision of the left eye, which had continued without much change.

Her vision was right eye $\frac{6}{7}$, left eye $\frac{6}{12}$. The right eye was normal except that the optic disk was slightly pale. In the left eye there was a suggestion of circumcorneal ciliary injection. The vitreous was cloudy. The optic papilla was hyperemic and blurred. At its lower temporal margin, above the course of the inferior temporal vein and crossed by the inferior temporal artery was an area, of about 1 disk diameter, of localized edema and exudate into and under the retina with more superficial edema extending nasally and into the macular region. There was a small hemorrhage into the retina overlying the inferior temporal artery (Fig. 15). On the arc perimeter and on the Lloyd stereocampimeter the visual field of the left eye showed a defect in the upper nasal quadrant continuous with the normal blind spot (Fig. 16).

The patient was moderately obese. She had mild chronic infectious arthritis, and periapical infection of five teeth. The tonsils were small and not definitely diseased. The Wassermann reaction of the blood was negative.

COMMENT

As Koehne concludes, no uniform cause has as yet been established for chorioretinitis juxtapapillaris. Granting the truth of Friedenwald's contention that the disease does not differ essentially from any other localized chorioretinitis, we would hardly expect the origin to be the same in all cases. In most of the published reports the cause has not been given. Zeeman believes that this is because cases of localized chorioretinitis occurring in association with some recognized gen-

USTRATING ARTICLE OF DR. HENRY P. WAGENER AND DR. JOHN F. GIPNER, "STUDIES OF THE VISUAL FIELDS IN LOCALIZED CHORIORETINITIS OF THE JENSEN TYPE."

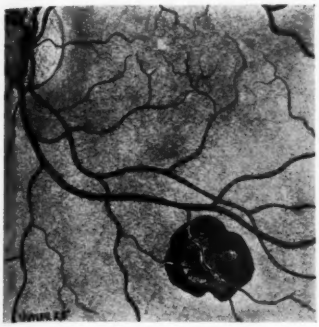


FIG. 13

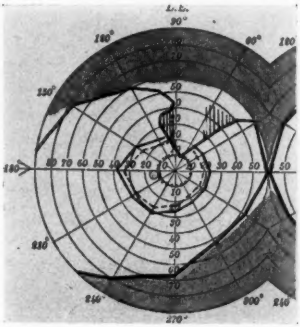


FIG. 14

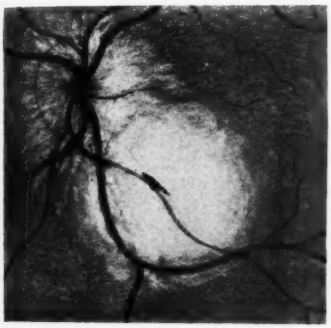


FIG. 15

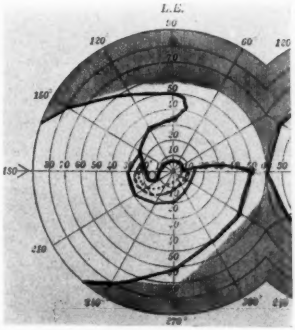


FIG. 16

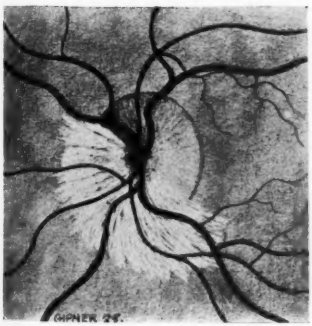


FIG. 17

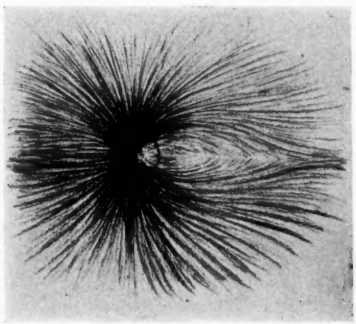


FIG. 18

eral disease such as syphilis or tuberculosis are reported under the heading of the general disease. Several of the published cases have been classified as ocular tuberculosis, mainly because of a positive general reaction to injections of tuberculin. In the light of our present knowledge of the effects of injections of nonspecific proteins, this can hardly be accepted as definite proof. The clinical course of chorioretinitis juxtapapillaris and of most cases of localized chorioretinitis is similar to that of other lesions of the eye due to foci of infection in various parts of the body. In most of the reported cases the search for foci has not been very thorough. In all of the cases in our series there were potential foci of infection. In Case 1 the acute choroiditis developed during the course of acute rhinitis and questionable nasal sinusitis. At the time of our examination this patient also had infected tonsils and an infected tooth. In Case 2 the tonsils had been removed because of chronic tonsillitis some time previous to our examination and there was marked pyorrhœa alveolaris. In Case 3 it is possible that the cervix uteri, the appendix, and the tonsils had been the foci. In Case 4 the tonsils and an infected kidney may have been sources of infection. In Case 5 there were infected tonsils. In Case 6 oral sepsis was present. In Case 7 there was a mild chronic infectious arthritis and five teeth showed periapical infection. As yet, however, no direct connection has been established clinically or experimentally between a focus of infection and chorioretinitis juxtapapillaris.

It is still unsettled whether the primary seat of the lesion in Jensen's disease is in the retina or in the choroid. Whenever there is a field defect, however, it is conceded that the retina must be injured; for, whenever the choroid alone is affected, there is no scotoma. If only the rod-and-cone layer of the retina is damaged, the scotoma will correspond in size, shape, and position with the defect seen ophthalmoscopically (Figs. 9 and 11). Most authors now agree that, in chorioretinal lesions, a sector-shaped field defect is produced by interference with the conducting power of the nerve fibers at the site of the lesion. For this reason, if the lesion is situated at the margin of the disk, the field defect will be continuous with the normal blind spot. If, however, as in Case 6, it is situated at some distance from the disk, the field defect will stop at

a corresponding point and will not be continuous with the normal blind spot. Jensen's original idea was that the scotoma was caused by interference with the blood supply to the periphery of the retina. In none of our cases were the arteries supplying the defective quadrant sufficiently sclerosed to cause the loss of vision. In Case 2 the retinal vessels that were mainly affected supplied the quadrant opposite the field defect. In Case 5, although only the superior nasal artery crossed the lesion, the resultant field defect included portions of both the inferior nasal and inferior temporal quadrants. In general the field defects in this disease do not correspond with those associated with evident thrombosis or embolism of individual branches of the central retinal artery. Jensen argued that the field defect could not be due to destruction of nerve fibers as there was no pallor of the corresponding sector of the papilla. This pallor, however, was present in Puiggari's third case and in Case 4 of the present series. Its absence in most cases is probably due, as Puiggari suggests, to the relatively small number of nerve fibers involved in the lesion.

STUDY OF THE FIELDS.

In attempting to estimate the course of the nerve fibers in the retina on the basis of the field defects produced by chorioretinal lesions at the margin of the disk, several points must be kept in mind. It is obviously impossible to establish a relationship in degrees between the fundus defect at the disk and the field defect at the periphery. It would be very difficult to make the fundus drawings exactly to scale, and no attempt was made to do this in our series. It is impossible to determine how much of the apparent lesion is really retinal and at just what point destruction of the nerve fibers has taken place. Also, it must be remembered that, especially in a large lesion, more fibers may be injured in the peripheral portion of the diseased area than at the margin of the disk.

For these reasons it is evident that comparison of the fundus picture with the field defect will not give a quantitative but a qualitative idea of the mode of radiation of the nerve fibers from the papilla into the retina. The size of the visual

field defect can be roughly estimated from the size of the fundus lesion, provided the visible scar is mainly retinal. This is demonstrated in Cases 3, 5, and 6, in which no choroidal vessels and no sclera were exposed. When atrophy of the choroid is marked, the extent of the destruction of the nerve fibers can not be made out accurately, and it is impossible to foretell the size of the field defect. In many cases, also, it is impossible to judge from the appearance of the lesion alone the exact point at which the nerve fibers are injured and to foretell the direction which the sector defect will assume. Thus one is compelled to consider that, in the perimetric fields the point of connection of the peripheral lesion with the normal blind spot indicates the portion of the margin of the disk at which the conducting power of the nerve fibers is destroyed.

In Case 1, although the apparent lesion affects the entire upper margin of the disk, the point of injury to the nerve fibers lies at the upper nasal angle. Here are found the strands of connective tissue which indicate the most severe involvement of the nerve-fiber layer. From the nature of the field defect, it can be deduced that of the fibers having their point of exit at the upper nasal angle of the disk, those situated more temporally radiate almost straight up whereas those situated more nasally tend to curve gradually to the upper nasal periphery and down toward the horizontal meridian, although the curve is not so sharp as is the case with the superior temporal and temporal fibers. Even the most curved of the fibers from the superior nasal angle do not meet the median raphé until near the limits of the functioning part of the retina, as represented on the average visual field.

In Case 2, although the lesion is again directly above the optic disk, it is evident that the point of injury to the nerve fibers lies more to the temporal half of the upper margin. It is apparent, from the field defect, that the upper temporal fibers curve more sharply toward the horizontal median line than do the upper nasal fibers and that all the fibers arising from this portion of the disk are curved. It is suggested from this case as well as from Case 5 that some of the fibers from the center of the upper margin of the disk and even from a spot slightly nasal to the center also tend to curve

temporally. This would be expected as rather necessitated by the large portion of the temporal margin occupied by the papillomacular bundle.

In Case 3, the lesion at the nerve margin, as evidenced by the proliferation of the connective tissue, can be assumed to involve most of the nasal margin; and the size of the connection of the sector defect with the normal blind spot supports this assumption. It is apparent from the field defect that the nasal fibers run radially toward the periphery. The defects in the upper and lower temporal fields in this case can be explained by assuming that some of the nerve fibers curving toward the horizontal meridian from the lower and upper nasal margins of the disk were caught in the main chorioretinal lesion at some distance peripherally from the disk. If this explanation can not be accepted, one is compelled to assume that the nasal fibers curve sharply upward and downward toward the vertical meridian. From the work of others and from the other fields in this series, this seems very improbable. It will be observed that in this case there was a restriction of the lower field of vision in each eye for which there was no adequate explanation.

In Case 4, a large lesion involves the entire lower margin of the disk. Pallor extends over the lower two-thirds of the disk, but that the actual destruction of the conducting power of the nerve fibers is limited to those leaving the lower third is shown not only by the actual size of the chorioretinal patch but also by the partial preservation of the upper fibers of the papillomacular bundle and by the preservation of the corresponding fibers radiating from the nasal margin of the disk. The connection of the field defect with the normal blind spot is also in correspondence with the smaller lesion. From the resultant field defect, one can appreciate the sharp curve of the fibers from the lower temporal disk margin ending in the median raphé; the more gradual curve of fibers from the lower margin to the inferior temporal retina; the radiating course of other fibers from the lower margin to the lower retina, and the gradual curve of the fibers from the lower nasal quadrant to the inferior nasal and peripheral nasal retina. It is also suggested that the fibers coursing from the nasal margin of the disk do not have a straight radial direc-

tion but tend to curve very slightly toward the horizontal meridian.

In Case 5, the nerve fibers are injured at the upper nasal margin of the disk. Here again the resultant field defect makes it evident that some fibers arising from the center of the upper margin of the disk tend to curve temporally, although not sufficiently to reach the median raphé. Fibers from the nasal half of the upper margin radiate to the upper periphery of the retina and perhaps slightly temporally, and, as the nasal margin is approached, the fibers tend to curve nasally although not so rapidly and sharply as in the case of the superior temporal fibers.

In Case 6, the lesion is situated about 2.5 disk diameters from the margin of the disk in the inferior temporal field. The nerve fibers involved must be a part of the bundles curving from the lower margin of the disk to the lower temporal retina. From the nature of the field defect, it is evident that in these bundles the fibers situated more peripherally tend to fan out temporally and thus fail to reach the median raphé within the limits of that part of the retina corresponding to the average field of vision.

In Case 7, it is difficult to determine the exact point of injury to the nerve fibers because the lesion is in the acute stage. Apparently, however, the injury is at the inferior temporal margin of the disk and involves the lowermost of the fibers curving toward the median raphé and also those fanning out to the lower temporal periphery of the retina. The defect in the field for 20 degrees immediately above and slightly temporal to the blind spot is probably due to edema involving the rod-and-cone layer of the retina, and not interfering with the conducting power of the nerve fibers. The preservation of the upper periphery of the field to a point slightly beyond the 90 degree meridian apparently indicates that at least some of the fibers radiating downward from the lower margin of the disk are not interfered with, and it seems probable that vision will be restored in that portion of the field between the blind spot and the upper preserved notch. The type of the residual field defect seen in Case 2 also suggests the possibility of such a return of vision. In view of the fact that edema was indubitably present when the field

was plotted in Case 7, it hardly seems rational to interpret the appearance of this field as indicating a still greater temporal curving of the fibers than is shown in the other fields in this series.

By a composite study of the field defects in this series of cases, it is possible to obtain the apparent direction of radiation of the nerve fibers from all portions of the disk margin. A schematic drawing of the direction of radiation as suggested by our cases is presented in Figure 17. It will be seen that this scheme agrees, in the main, with the diagrams presented by Elliott in his textbook on glaucoma, and with his statements on the subject. Our cases, however, indicate that there is not an even division into nasal and temporal halves of the fibers radiating from the upper and lower disk margins, but that the temporally curving fibers occupy definitely more than half the rim of the papilla. That this is true can readily be seen in cases in which the radiation of the nerve fibers from the disk is visible because of their medullation. A typical case is pictured in Figure 18.

The fanning out temporally and below of the more peripheral fibers of the group curving temporally toward the median raphé is suggested in our fields. This feature is not well represented in Elliott's diagrams, although it is mentioned by him in the text. It is very definitely suggested in the drawing of the medullated fibers in Lang's case.

In all other points the method of radiation of the nerve fibers suggested by the field defects in our series of cases agrees closely with the generally accepted ideas.

A curved scotoma such as is described by van der Hoeve and Groes-Petersen was not found in any of the cases in our series, although the field defect in Case 2 resembles it in some respects. There was nothing in our fields to support van der Hoeve's views on the method of supply of the retina adjoining the median raphé temporal to the fovea.

BIBLIOGRAPHY.

1. Elliott, R. H. *Glaucoma.* London, H. H. Lewis and Co., 1918, 188–193.
2. Friedenwald, Harry. "Relation of chorioretinitis juxtapapillaris (Jensen's disease) to other forms of chorioretinitis." *Tr. Ophth. Soc. U. Kingdom,* 1924, xliv., 308–326.

3. HOEVE, J. VAN DER "Nervenfasern Defekte bei Retino-choroiditis juxtapapillaris (Edmund Jensen)". *Klin. Monatsbl. f. Augenh.*, 1914, liii., 487–493.
4. JENSEN, EDMUND. "Retino-choroiditis juxtapapillaris." *Arch. f. Ophth.*, 1908–1909, lxix., 41–48.
5. KOEHNE, W. "Zur Aetiology der Retinochoroiditis (E. Jensen)". *Klin. Monatsbl. f. Augenh.*, 1920, lxv., 882–887.
6. LANG, B. T. "A case of opaque nerve fibers." *Tr. Ophth. Soc. U. Kingdom*, 1920, xl., 178–179.
7. MOORE, R. F. *Medical Ophthalmology.* Philadelphia, Blakiston, 1925, p. 282.
8. PUIGGARI, M. I. "Retino-choroiditis juxtapapillaris de Jensen." *La Prenza Medica Argentina*, 1917, iv., 30–32.
9. ZEEMAN, W. P. C. "Der Adaptation in einem Falle luetischer Neuritis retinae." *Arch. f. Ophth.*, 1921, cvi., 1–9.

THE ACTION OF ATROPINE IN OCULAR INFLAMMATIONS.[1]

By Dr. FRANCIS HEED ADLER.

From the Department of Physiology, University of Pennsylvania Medical School.

(*With two illustrations in the text.*)

INTRODUCTION.

ATROPINE is the sheet anchor in the treatment of most inflammatory diseases of the eye. There is no doubt of its efficiency in the treatment of inflammations of the whole anterior segment, especially in those diseases affecting the iris and ciliary body. If one inquires how atropine exerts its beneficial action the answer found in text books of ophthalmology or pharmacology is that it paralyzes the endings of the 3rd Nerve and thereby puts the ciliary and sphincter muscles at rest and dilates the pupil. The modus operandi of atropine action is therefore put on a purely mechanical basis—its use in these terms implies the acquisition of anatomic and physiologic rest. Clinical experience teaches us that the dilatation of the pupil in itself is an important factor, for the formation of posterior synechia is thereby prevented in large measure, and any fresh synechia which have formed are broken up. At the same time cases are often seen in which the formation of synechia is a rare occurrence, as in interstitial keratitis, phlyctenular kerato-conjunctivitis, and the various forms of corneal inflammations and dystrophies associated with the formation of deposits on the corneal endothelium (keratic precipitates). In these cases atropine is also of decided benefit.

[1] Read before the Section on Ophthalmology, College of Physicians of Philadelphia, April 15, 1926.

This raises the question whether there is another action of atropine on the eye besides those mentioned.

In the course of some experiments previously published (1) it was found that eserine caused a marked increase in the protein content of aqueous humor when instilled into the conjunctival cul de sac, and that if the eye had been previously atropinized eserine no longer caused such an increase in the protein content. This had been shown previously by Wessely (2) and later by Seidel (3) who had claimed from such experiments proof of the secretory nature of the ciliary epithelium. Our experiments indicated however that eserine did not increase the protein content of aqueous by stimulating gland cells to secrete, as claimed by Seidel (3), for the same increase in protein occurred with eserine after complete destruction of the 3rd Nerve endings to the entire eye. It looked as though eserine had a direct action on blood vessels, causing them to become more permeable and to leak protein into the aqueous. This hypothesis was therefore made, and it carried with it as a corollary the assumption that atropine must have an opposite action, *i.e.*, it must have a direct action on blood vessels, making them less permeable to protein molecules.

If this hypothesis is correct it offers a satisfactory explanation of one of the effects of atropine in ocular inflammations, for one of the cardinal signs of all inflammatory processes is a leakage of protein from blood vessels. Any drug which tends to prevent such a leakage will allay inflammation. It is conceivable that although inflammation itself is a protective bodily mechanism, the consequences of the inflammatory process are detrimental and the accumulation of a protein rich transudate in the tissues or in serous cavities means long drawn out processes of absorption and repair.

It was the purpose of these experiments therefore to test this hypothesis.

METHODS.

It was necessary to find some means of producing an increase in the protein content of aqueous humor which would be fairly constant and which could be altered so that the intensity of the effect could be controlled. Trauma to the

globe, artificially produced corneal ulcers, etc., could not be considered because of the inability to control the intensity of their action.

The following group of drugs was therefore used, and the effects of each will be described separately.

1. *Dionin.*—This drug in 5%–10% solution dropped into the conjunctival cul de sac produces an inflammatory reaction, with dilatation of the finer arterioles and capillaries, and an outpouring of blood serum, so that the conjunctiva becomes œdematous.

2. *Subconjunctival injections of hypertonic salt solution.*—If 0.2*cc* 10% Na Cl solution be injected under the bulbar conjunctiva there will appear in the course of half an hour a marked œdema of the whole conjunctiva due to the attraction of fluid out of the blood vessels partly due perhaps to the hypertonicity of the salt solution, and partly due to the irritant action of the salt on the blood vessels, dilating them.

3. *Histamine.*—This drug dilates capillaries under certain conditions, and causes an œdema of the part to which it is applied. Local application in the unanæsthetized cat, however, failed to evoke any œdema, and no constant change in the protein content when injected intravenously in an anæsthetized animal. No change in blood pressure occurred with an intravenous injection of histamine unless the animal were under deep anæsthesia. The effects of this drug therefore were too uncertain and its use was discontinued.

4. *Oil of Mustard.*—This drug causes a violent inflammatory reaction when dropped in the eye, with the appearance of a tremendous œdema. It can be diluted however in lard oil, and any desired intensity of effect obtained. The œdema of the conjunctiva so produced has been studied by Bruce (4), who obtained evidence of the local axone—reflex nature of an inflammatory œdema so produced. Hirschfelder (5) and others have also used this drug in studying the nature of localized œdema.

METHODS USED IN DETERMINING THE PROTEIN CONTENT OF AQUEOUS.

Normal aqueous humor contains approximately 0.025% Protein. The most satisfactory method for quantitative

determination of dilute protein solutions was found to be the nephelometric method described by Dennis and Ayer (6) for the determination of protein in cerebro-spinal fluid.

This was the method used in the experiments cited in the previous paper (1). Several modifications have been made to insure greater accuracy in the method. Only 0.2*cc* of aqueous was used instead of 0.4*cc*. It was believed that occasionally the withdrawal of nearly 0.5*cc* of aqueous might rupture the blood vessels and thereby lead to a high protein content in normal fluid. By reducing the quantity used to 0.2*cc* it was only necessary to withdraw 0.3*cc* as a maximum. The standard solutions made up from normal blood serum were increased in number and made according to the system of standards adopted by Dreyer (7) as follows:—

Standards:—1.0%, 0.08%, 0.06%, 0.05%, 0.04%, 0.035%, 0.03%, 0.025%, 0.02%, 0.015%.

It was found that greater accuracy could be obtained by reading the unknown against a picked standard and then checking against the standard nearest in protein per cent to the unknown so calculated. The standard should not vary more than 30% from the unknown for accurate readings. 1*cc* of double glass distilled water was introduced into a 10*cc* volumetric flask. 0.02*cc* of aqueous humor was added to this by means of the calibrated glass pipettes described in the previous paper. (1) 1*cc* of a 5% solution of sulphosalicylic acid was then added drop by drop while the tube was being gently shaken. This was allowed to stand ten minutes and then distilled water added to the 10*cc* mark. The standard was made up the same way. After shaking, the standard and unknown were poured into the nephelometer tubes and read in a Klett nephelometer in the dark room. Before each experiment the nephelometer tubes were filled with distilled water and examined to see that the fields were nearly dark. The tubes and plungers must be scrupulously clean, and in order to prevent scratching or deposition of dust must only be touched with fine crepe-de-chine cloth. Between experiments the nephelometer tubes and volumetric flasks are kept immersed in strong cleaning fluid and are thoroughly rinsed in tap water followed by distilled water. With this

method unknown solutions of about 0.02% protein can be determined with an error of two to three per cent.

EXPERIMENTS.

1. *The Difference in Protein Content of the Two Normal Eyes.*

Cats were used in all experiments. Aqueous was withdrawn simultaneously from each eye under ether anæsthesia. On page 274 of the previous article (1) a table was given showing the difference in protein content between the two eyes of six cats; these figures are included in the present table, which has been considerably increased. With the exception of one 48% difference the figures all lie under 13%. Including this one high figure the average difference between normal eyes is 7.8%, or omitting this the difference is 5.2%. In the experiments to follow, however, the difference between the control and the experimental eye should be at least 10% in order to be regarded as significant.

TABLE I.

THE DIFFERENCE IN PROTEIN CONTENT OF THE TWO NORMAL EYES WHEN THE AQUEOUS IS WITHDRAWN SIMULTANEOUSLY.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*
2/23/24	1	0.0278%	0.0274%	1.5%
3/11/24	2	0.0192	0.0189	1.5
12/17/24	3	0.0387	0.0389	0.5
12/17/24	4	0.0317	0.0314	0.9
12/18/24	5	0.0303	0.0314	3.6
12/19/24	6	0.0377	0.0429	13.
1/26/25	7	0.0458	0.0507	9.6
5/ 1/25	8	0.0301	0.0448	48.
5/ 6/25	9	0.0254	0.0262	3.3
5/ 7/25	10	0.0529	0.0551	4.1
7/23/25	11	0.0388	0.0342	12.
7/23/25	12	0.0427	0.0442	3.
7/24/25	13	0.0396	0.0421	6.
7/24/25	14	0.0434	0.0432	0.4
11/11/25	15	0.0394	0.0441	11.
11/26/25	16	0.0310	0.0335	8.
11/27/25	17	0.0198	0.0212	7.

As a further control for the experiments to follow, aqueous was withdrawn from one eye and, after an interval of one

hour aqueous from the second eye was withdrawn. This necessitated two etherizations. In order to minimize the effect of the anæsthesia as much as possible, asphyxia was carefully avoided and the anæsthesia was not pushed to the extent of disappearance of the corneal reflex.

TABLE II.

THE DIFFERENCE IN PROTEIN CONTENT OF THE TWO NORMAL EYES WHEN THE AQUEOUS IS WITHDRAWN FROM EACH EYE ONE HOUR APART.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
4/28/25	1	0.0451%	0.0559%	−19. %	O. D. withdrawn last.
4/28/25	2	0.0438	0.0547	−18.	" " "
5/ 8/25	3	0.0367	0.0379	−3.2	" " "
5/ 8/25	4	0.0406	0.0416	−2.4	" " "
5/ 8/25	5	0.0344	0.0382	−9.9	" " "
9/ 9/25	6	0.0386	0.0375	+2.9	" " "
9/ 9/25	7	0.0379	0.0384	−1.3	" " "
9/ 9/25	8	0.0288	0.0274	+5.	" " "

In two of eight experiments the second aqueous was considerably lower than that from the first eye. These two experiments led to more rigid precautions being taken regarding the depth of anæsthesia and in the remaining experiments the two etherizations were made as nearly alike as possible. In none of these is there any significant change. It seems evident then that if the state of anæsthesia be kept fairly constant the withdrawal of aqueous from the second eye one hour after the first has been withdrawn does not affect the normal difference in the protein content between the two eyes.

2. *The Influence of Atropine on the Increase in Protein Caused by Dionin.*

1 drop 5% dionin is instilled into the right eye at half hour intervals for 1 to 5 drops. One half hour later the cat is etherized and the aqueous withdrawn from both eyes.

This table shows an increase in the protein content of aqueous due to the influence of dionin. The results are not strik-

TABLE III.

THE DIFFERENCE IN PROTEIN CONTENT OF THE TWO EYES WHEN DIONIN HAS BEEN INSTILLED INTO THE RIGHT EYE.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
1/2/25	1	0.0334%	0.0350%	−4.5%	1 drop 5% dionin, aqueous withdrawn one half hour later. No chemosis.
1/2/25	2	0.0346	0.0321	+7.8	2 drops 5% dionin, aqueous withdrawn half hour later. Definite chemosis.
1/5/25	3	0.0392	0.0231	+69.	3 drops 5% dionin, aqueous withdrawn half hour later. Definite chemosis.
1/5/25	4	0.0552	0.0549	+0.5	4 drops 5% dionin, aqueous withdrawn half hour later. No chemosis.
1/7/25	5	0.0490	0.0376	+30.3	2 drops 5% dionin, aqueous withdrawn 1 hour later. Definite chemosis.
1/7/25	6	0.0320	0.0301	+6.3	3 drops 5% dionin, aqueous withdrawn half hour later. No chemosis.
1/9/25	7	0.0209	0.0198	+5.5	4 drops 5% dionin, aqueous withdrawn half hour later. Very slight chemosis.
1/19/25	8	0.0366	0.0358	+2.2	5 drops 5% dionin, aqueous withdrawn 15 minutes later. No chemosis.
1/30/25	9	0.0347	0.0236	+42.8	2 drops 10% dionin, aqueous withdrawn half hour later. Definite chemosis.
7/24/25	10	0.0549	0.0495	+10.	3 drops 10% dionin, aqueous withdrawn half hour later. Definite chemosis.
7/27/25	11	0.0946	0.0409	+46.	3 drops 5% dionin, aqueous withdrawn half hour later. Definite chemosis.

ing, there being only five very definite positive results, the other seven all lie within what might be considered the normal difference, (compare Table I). The striking regularity with which the dionin eye is higher than the control, however slight, is of significance, and this, coupled with the four very positive results, leads us to the conclusion that dionin is an effective drug in producing an increase in the protein content: In every case where there was a marked increase in the protein of the aqueous there was also a very definite chemosis of the conjunctiva. In four cases there was no chemosis at all and in none of these was there any increase in the protein of the aqueous humor. This suggests that the increase in protein is to be looked upon as a pure transudate from the intraocular blood vessels, produced in the same way as the transudate from the conjunctival vessels causing the œdema.

TABLE IV.

THE EFFECT OF DIONIN ON THE RIGHT EYE AFTER ATROPINIZATION.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
1/ 9/25	1	0.0278%	0.0366%	−24. %	3 drops 5% dionin, aqueous withdrawn half hour later. No chemosis.
1/ 9/25	2	0.0303	0.0311	−2.2	4 drops 5% dionin, aqueous withdrawn half hour later. Definite chemosis.
1/12/25	3	0.0401	0.0406	−1.	4 drops 5% dionin, aqueous withdrawn half hour later. Definite chemosis.
1/19/25	4	0.0221	0.0200	+10.5	4 drops 5% dionin, aqueous withdrawn half hour later. Definite chemosis.
1/30/25	5	0.0263	0.0364	−27.7	2 drops 10% dionin, aqueous withdrawn half hour later. No chemosis.
7/27/25	6	0.0178	0.0186	−4.	4 drops 10% dionin, aqueous withdrawn half hour later. No chemosis.

Table IV gives the results of six experiments in which the right eye has been atropinized and then 2 to 4 drops of dionin instilled in exactly the same manner as the control series in Table III. One half hour after the last drop the cat is anæs-

TABLE V.

THE EFFECT OF DIONIN DROPPED INTO EACH EYE, THE RIGHT EYE HAVING BEEN PREVIOUSLY ATROPINIZED.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
7/28/25	1	0.0290%	0.0282%	+2.8%	4 drops 10% dionin, aqueous withdrawn half hour later.
7/28/25	2	0.0231	0.0233	−0.9	4 drops 10% dionin, aqueous withdrawn half hour later.
7/28/25	3	0.0296	0.0312	−5.	4 drops 10% dionin, aqueous withdrawn half hour later.
7/29/25	4	0.0168	0.0214	−21.	4 drops 10% dionin, aqueous withdrawn half hour later.
7/30/25	5	0.0234	0.0211	+10.	4 drops 10% dionin, aqueous withdrawn half hour later.

TABLE VI.

THE EFFECT OF DIONIN IN BOTH EYES, THE RIGHT EYE HAVING BEEN PREVIOUSLY ATROPINIZED. EXPERIMENTS CARRIED OUT IN SUCH A WAY THAT THE POSSIBILITY OF ATROPINIZATION OF THE LEFT EYE IS PREVENTED.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*
8/18/25	1	0.0288%	0.0409%	−29.3%
8/18/25	2	0.0201	0.0246	−18.2
8/19/25	3	0.0299	0.0386	−22.
8/19/25	4	0.0312	0.0358	−12.
8/21/25	5	0.0449	0.0314	+42.
8/21/25	6	0.0188	0.0227	−17.1
8/24/25	7	0.0451	0.0677	−33.
8/24/25	8	0.0165	0.0214	−22.
8/25/25	9	0.0289	0.0357	−19.
8/25/25	10	0.0213	0.0243	−12.

thetized and the aqueous withdrawn from both eyes. In no case was the protein content increased. In fact in two cases there was a very definite decrease in the eye with the atropine.

Table V shows the effect of dionin dropped into each eye, one of which has been previously atropinized. By dropping the

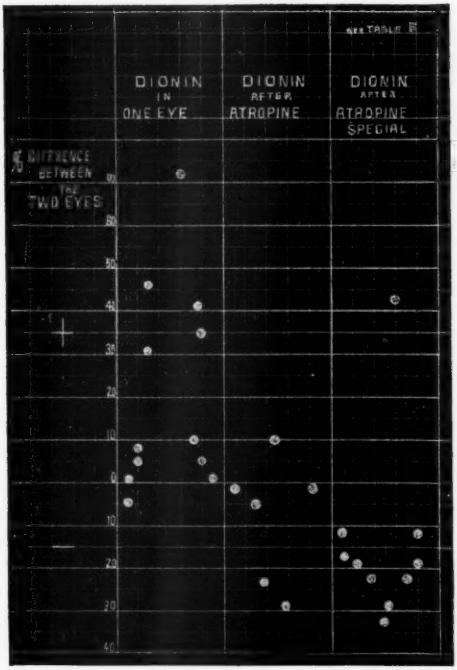


FIG. 1

Graph showing the effect of dionin on eyes with and without atropine. The normal differences lie between + and − 10.

dionin in both eyes it was expected that the difference between the two eyes would be more marked than in Table IV. This is not the case however, and it was noted that in several of the animals after atropinization, both eyes were dilated and inactive to light. It was felt, therefore, that sufficient atropine

might be absorbed into the circulation from one to atropinize the opposite eye. The following experiments were devised therefore, to eliminate the possibility of absorption of atropine and atropinization of the control eye.

2 drops of 5% dionin were instilled half an hour apart into the left eye. One half hour later the animal was anæsthetized and the aqueous from the left eye withdrawn and examined. Atropine was then started on the right eye,—one drop of 2% atropine sulphate being instilled every fifteen minutes for four drops. Two drops of 5% dionin were then instilled into the right eye in exactly the same manner as had been done in the left. One half hour after the last drop the animal was reanæsthetized and the aqueous from the right withdrawn and examined.

TABLE VII.

THE EFFECT OF 0.2cc OF 10% SODIUM CHLORIDE INJECTED SUBCONJUNCTIVELY IN THE RIGHT EYE.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*
2/24/25	1	0.0300%	0.0373%	−19.%
3/ 9/25	2	0.100	0.0335	+198.
3/ 9/25	3	0.0787	0.0197	+299.
3/13/25	4	0.0322	0.0349	−7.7
3/13/25	5	0.0714	0.0471	+51.
3/16/25	6	0.0462	0.0432	+6.9
3/16/25	7	0.0826	0.0297	+177.
3/16/25	8	0.0540	0.0444	+21.
3/16/25	9	0.0515	0.0429	+20.
3/18/25	10	0.0862	0.0487	+77.
4/ 6/25	11	0.1410	0.0329	+328.
4/ 6/25	12	0.1310	0.0390	+236.
4/20/25	13	0.1013	0.0660	+53.
4/17/25	14	0.0485	0.0453	+7.
4/17/25	15	0.0898	0.0903	−0.5
8/14/25	16	0.0177	0.0172	+2.9
8/14/25	17	0.0213	0.0235	−9.3

Table VI gives the results of ten experiments carried out in this manner. In all save one experiment the atropinized eye was very much lower in protein content than the unatropinized. This method brought out very strikingly the fact that atropine prevents the increase in protein due to dionin.

3. *The Effect of Atropine on Sub-Conjunctival Injections of Hypertonic Salt Solution.*

Table VII shows the effect of an injection of 0.2*cc* of 10% sodium chloride solution subconjunctivally in the right eye. One hour after the injection the cats were etherized and the aqueous withdrawn from both eyes. In 10 out of 17 experiments there was a very definite increase in the protein content.

TABLE VIII.

THE EFFECT OF 0.2CC OF 10% SODIUM CHLORIDE INJECTED SUB-CONJUNCTIVALLY IN THE RIGHT EYE AFTER ATROPINIZATION.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
3/13/24	1	0.0546%	0.0578%	−5.5%	
3/20/25	2	0.0483	0.0485	−0.4	
3/20/25	3	0.0444	0.0495	−10.3	
3/20/25	4	0.0409	0.0440	−7.0	
3/11/25	5	0.0944	0.0373	+153.	
3/23/25	6	0.1176	0.0260	+352.	
3/23/25	7	0.0704	0.0510	+38.	
3/23/25	8	0.1111	0.0714	+55.	
3/23/25	9	0.0571	0.0591	−3.4	
3/23/25	10	0.0406	0.0520	−21.9	
4/ 7/25	11	0.1081	0.0265	+307.	
4/20/25	12	0.0258	0.0215	+20.	
5/11/25	13	0.0267	0.0245	+8.9	Salt injected both eyes.
5/11/25	14	0.0297	0.0340	−12.	Salt injected both eyes.
5/15/25	15	0.0302	0.0412	−26.	Salt injected both eyes.
5/16/25	16	0.0277	0.0289	−4.1	Salt injected both eyes.
5/18/25	17	0.0412	0.0291	+29.	Salt injected both eyes.

Table VIII shows a similar series of experiments in which the right eye was thoroughly atropinized before the salt solution was injected. One hour after the injection the cats were etherized and the aqueous withdrawn from both eyes. In 7 out of 17 experiments there was a definite increase in the protein content. These experiments failed to show the effect of atropine in inhibiting the increase in protein due to sub-

conjunctival injections of hypertonic saline. It will be noticed, however, that in experiments No. 13 to 17 inclusive the salt was injected into both eyes in order to determine whether it was actually increasing the protein content in the control eye. In none of these is there an increase. This coincides with the marked variability to be found in the control series. When the salt solution is effective in increasing the protein content, it does so to such an extent that the effect of atropine probably cannot be demonstrated experimentally.

4. *The Effect of Atropine on the Action of Oil of Mustard.*

If one drop of 0.2 per cent. oil of mustard in lard oil be instilled into the conjunctival cul-de-sac a marked œdema usually results and the protein content after one hour is found to be well increased.

TABLE IX.

THE DIFFERENCE IN PROTEIN CONTENT BETWEEN THE TWO EYES WHEN 1 DROP OF 0.02% OIL OF MUSTARD IS INSTILLED IN EACH EYE ONE HOUR APART.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
12/21/25	1	0.350 %	0.398 %	+13%	O. S. withdrawn last.
12/21/25	2	0.0661	0.104	+57	" " "
12/28/25	3	0.0304	0.0322	+6	" " "
12/28/25	4	0.0285	0.0392	+37	" " "
12/28/25	5	0.0722	0.0295	−59	" " "
12/28/25	6	0.450	0.113	−72	" " "
1/ 8/26	7	0.0603	0.0745	+23	" " "
1/11/26	8	0.0231	0.0232	0	" " "
1/11/26	9	0.0532	0.0975	+83	" " "
1/11/26	10	0.261	0.740	+183	" " "
1/11/26	11	0.0488	0.0669	+37	" " "

Table IX shows the effects of one drop of 0.2% oil of mustard instilled into the right eye and the aqueous withdrawn one hour later. One drop is then instilled into the left eye and the aqueous withdrawn one hour later. In all of the experiments with oil of mustard the cats were anæsthetized by an intraperitoneal injection of Barbitol-Sodium (0.4 Grm. per kilo. body weight) before the mustard was instilled. This

was done for two reasons. It was thought that some of the discrepant results obtained in the previous experiments might be due to variations in depth of anæsthesia and secondly it was found that oil of mustard caused too much discomfort to be used without constant anæsthesia. In seven out of eleven experiments there is a definite increase in the protein content of the second eye. It is difficult to explain why this should occur. It is analogous to results previously obtained by a number of German workers (8) (9) (10) who tried to show that trauma inflicted on one eye caused changes in the opposite eye. They believed that sympathetic ophthalmia might be explained on this basis. Wessely (11) and other workers were unable to confirm their results. Although these experiments might be interpreted in this manner, we have no other evidence to offer that this is actually the case. In the experiments done in which one drop of oil of mustard was instilled into one eye and the aqueous withdrawn from both eyes one hour later, we never found the aqueous of the control eye to contain more than a normal amount of protein. It is possible, however, that trauma to the first eye makes the second eye more vulnerable so that similar trauma to this eye is now more effective than in the first eye. We cannot explain the marked decrease in the second eye shown in experiments 5 and 6.

TABLE X.

THE DIFFERENCE IN PROTEIN CONTENT BETWEEN THE TWO EYES WHEN THE LEFT EYE HAS BEEN ATROPINIZED AND THEN ONE DROP OF 0.02% OIL OF MUSTARD DROPPED INTO EACH EYE AN HOUR APART.

Date	*Exp. No.*	*O. D.*	*O. S.*	*Difference*	*Remarks*
12/18/25	1	0.492 %	0.125 %	−74. %	C. S. withdrawn last.
12/19/25	2	0.0985	0.0422	−57.	" " "
12/22/25	3	0.0285	0.0318	+11.	" " "
12/28/25	4	0.1321	0.0920	−30.	" " "
12/29/25	5	0.0952	0.1057	+11.	" " "
1/ 4/26	6	0.155	0.117	−24.	" " "
1/22/26	7	0.0307	0.0297	−3.2	" " "
1/25/26	8	0.175	0.055	−70.	" " "

Table X gives the results in a similar series of experiments in which the left eye was atropinized before the instillation of oil of mustard. In 5 out of 8 experiments there was a marked decrease in the protein content of the atropinized eye as con-

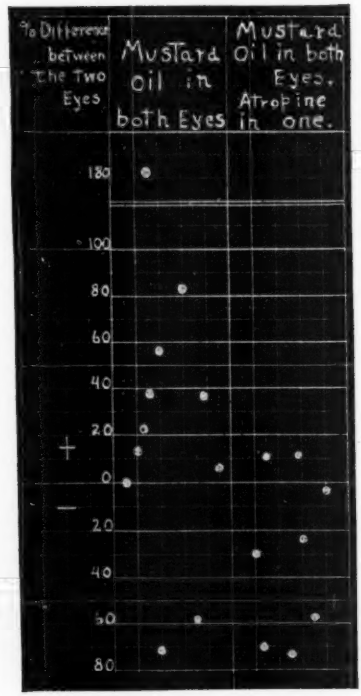


FIG. 2

Graph showing the effect of oil of mustard in both eyes and the effect where one eye has been previously atropinized.

trasted with the unatropinized eye. In no experiment was this eye materially higher than the first. Contrasting these results with those of the control series in Table IX, there can be no doubt that atropine successfully prevents the marked increase in protein caused by oil of mustard.

SUMMARY.

Experiments were devised to determine whether or not atropine had any effect on the permeability of the ocular blood vessels. From some work previously published it was thought there was some evidence to support the hypothesis that atropine lessened the amount of protein in the aqueous humor caused by eserine by a direct action on blood vessels.

A group of drugs were selected which caused an inflammatory transudate from the intraocular blood vessels—these drugs were used with and without a previous atropinization of the eye. It was found that atropine diminished, or prevented altogether, the action of dionin and mustard oil in increasing the protein content of aqueous. This effect was not clearly shown when subconjunctival injections of hypertonic salt solution were used, but this was probably due to the fact that this method of increasing the protein content cannot be well controlled. There is either no increase at all or such a marked outpouring of blood serum that the atropine fails to check it.

The experimental results obtained are not all that could be desired. There are occasional marked discrepancies in the results, which it was not found possible to control. If one considers, however, how many variable factors must exist which cannot be controlled the experimental data is fair evidence of a direct action of atropine on blood vessels. The chief objection to such conclusions being drawn is probably to be found in the claim of Mawas (12) who believed that the increased protein content occurring after instillation of eserine was due to the constriction of the pupil. He considered that in this state the iris vessels were exposed and therefore leaked protein when the aqueous was withdrawn, more readily than when the iris was dilated. This is incorrect, however, for in most of our experiments the pupils on the two sides were equal after the instillation of mustard oil on one side for example, and yet this side showed a very much higher protein content. Further, we have studied the effect of the size of the pupil on the protein content by withdrawing the aqueous from one eye in a semi-dark room so that the pupil was dilated, and then exposing the opposite eye to bright light and with-

drawing the aqueous when the pupil was constricted. We found no difference between the two sides.

Within the last few months Burn (13) has published a paper proving by perfusion experiments that pilocarpine dilates capillaries. He did not, unfortunately, test out the antagonistic action of atropine.

Our experiments give evidence that atropine has such an antagonistic action, but similar experiments to those of Burn must be carried out before this action is accepted as proved.

In carrying out this work many helpful suggestions and criticisms have been obtained from Prof. A. N. Richards of the Dept. of Pharmacology and from Prof. H. C. Bazett of the Dept. of Physiology; for which we are duly grateful. In part of this work we have been ably assisted by Miss Juliana Tatum.

CONCLUSIONS.

Atropine has a direct action on the blood vessels in the eye decreasing their permeability to protein. When the eye is inflamed for example by the instillation of dionin or oil of mustard the protein content of the aqueous humor is increased, but if the eye has been previously atropinized the increase in protein is either partially checked or altogether prevented. It is suggested that this action of atropine is one of the beneficial effects of this drug in the treatment of ocular inflammations.

BIBLIOGRAPHY.

1. ADLER, F. H. and LANDIS, E. M. *Archives of Ophthalmology*, vol. liv., 1925, p. 265.
2. WESSELY, K. *Centralbl. f. praktisch. Augenheilk.*, vol. 37, 1918, p. 303.
3. SEIDEL, E. *Graefe's Arch. f. Ophth.*, vol. 102, 1920.
4. BRUCE, N. *Quarterly Journ. Exp. Phys.*, vol. 6, 1913, p. 339.
5. HIRSCHFELDER, A. D. *American Journ. Phys.*, vol. 70, 1924, p. 507.
6. DENNIS and AYER. *Arch. of Internal. Med.*, vol. 26, 1920, p. 436.
7. DREYER, G. and GARDNER, A. *Biochem. Journ.*, vol. 10, 1916, p. 399.
8. GRUNHAGEN and JESNER. *Centralbl. f. Augenheilk.*, vol. 4, 1880.
9. JESNER. *Pfluger's Arch. f. d. ges. Phys.*, vol. 23.
10. MOOREN and RUMPF. *Centralbl. f. Med. Wissenschaft*, vol. 19, 1880.
11. WESSELY. *Graefe's Arch. f. Ophth.*, vol. 50, 1900, p. 123.
12. MAWAS, J. *Rec. d'Opht.*, vol. 33, 1911, p. 13.
13. BURN, J. H. *British Journ. Phys.*, October, 1925.

REPORT OF THE ANNUAL CONGRESS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By Mr. H. DICKINSON, London.

The Society's Annual Congress was held on April 29th and 30th and May 1st, under the presidency of Sir John Herbert Parsons, C. B. E., D. Sc., F. R. S.

The Presidential Address.

The President, after thanking members for the honor they had shown him, delivered an address on the progress of ophthalmology, and the part which the Society had taken in that progress. Biological advances must of necessity lag behind those of physics and chemistry, but medicine was now gradually emerging from the dominance of empiricism and metaphysical speculation, though it could never be expected to rank as a pure science. It was natural to attempt short-cuts to cure, but there was no royal road to scientific certitude. Still, man's eagerness to get well kept him still under the subtle witchery of the quack and the charlatan. The Society must continue to wage war against this quackery, but it must be done without feelings of self-glorification, for in the practice of medicine there was an empiricism which was legitimate. The view taken of ophthalmology was often too narrow; it did not sufficiently take note of the ways in which the subject impinged on public life, nor how advances in other fields could be applied to this.

Among the problems concerned with improved conditions for the worker were those of illumination and fatigue; the effect of flickering lights; the legibility of types, light therapy.

He appealed to members to encourage their clinical assistants to equip themselves to carry on research work in the specialty. He made special reference to light therapy, which was practised largely at the present time, and often by persons who did not sufficiently realize how potent an agent they were employing.

SIR JOHN said there existed the idea that ophthalmology was a narrow, sharply delimited field of work, but the eye, as an important receptor organ, could only be adequately studied as an integral part of that varied receptor apparatus which was the gateway of perception. Adequate stimuli to the eye included wave-lengths some of which possessed properties which were deleterious to living tissues, such as those of the retina.

On the ethical side, the President said it was lamentable that the pretentions of a relatively small yet influential group of sight-testing opticians should prevent ophthalmologists taking part in a collaboration with those who were devoting all their energies to optical research. Still he was hopeful of a solution of the sight-testing optician problem.

The hand of specialization, he declared, was isolation, and coöperation was the cure of this. He expressed the hope that the Society would continue to contribute worthily to the repair and improvement of the great edifice which, erected by their predecessors, had been handed down to the present members.

On the proposition of MR. J. H. FISHER, seconded by DR. G. MACKAY, the President was cordially thanked for his address.

OBSERVATIONS ON COLOR VISION.

PROF. H. E. ROAF gave a careful disquisition on this subject, aided by slides. He said he had been interested in it from the bio-chemical point of view, and thought an enquiry as to the way in which radiant energy could be transformed into nerve impulses would be useful. In what way, he asked, was color vision related to the wave length of light? The manner in which the text-books of physiology treated the subject was brief and not very satisfactory. It was neces-

sary, he thought, to look for some sort of transformer in the retina itself. He got a color-blind person to copy a color diagram, using a box of ordinary colors. He found that when looking through a blue glass the normal person made the same mistakes as a color-blind person did. In 28 cases the defect was always in the red end of the spectrum: the red rays were not distinguished from the rays from other parts of the spectrum. On cutting out the red end of the spectrum most of the mistakes ceased. He found that the sensitivity of the color-blind person to the red end of the spectrum was the same as that of the normal person. In making the tests he found it necessary to render the intensity of the light used the same for all parts of the spectrum, because without such adjustment the degree of luminosity formed a good guide to the examinee. He thought the differentiating mechanism was likely to reside in the cones. In the cones of birds, reptiles and amphibians, there were colored globules, namely, at the junction of the cone with the cone body. This was known to Helmholtz, and was mentioned by him as a possible explanation of color vision. If color globules could be found in the human eye, these would afford an explanation of many of the occurrences in color vision. In conclusion he dealt with the question of visual fatigue.

THE PRESIDENT spoke of the variations noticed in the shade of color produced in text-books, depending on the number of copies which had already been struck off. It was evident, he said, that in all varieties of color-blindness there were considerable ranges, and the tester could get only a rough idea of what the patient really saw. He was glad to hear Prof. Roaf's good word for the trichromatic theory as it was a valuable one. A hypothesis was valuable only in its capacity to explain known facts and to originate new lines of enquiry.

THE VALUE OF COLLECTIVE DRAWINGS.

MR. RAYNER D. BATTEN read a paper bearing the title "The value of collective drawings of diseases of the fundus as an aid to research." He showed a large series of drawings, and several speakers supported his views.

Intra- and Extra-Ocular Melanomata.

On Thursday afternoon a discussion took place on this subject, and was opened by Mr. W. G. Spencer, a general surgeon. He said that melanomata, pigmented nævi or moles, were either developmental defects, or acquired new formations which, though for a time remaining benign, underwent gradual changes which ended in malignancy. Melanin was now recognized as being directly connected with a disturbance of cell matabolism. Melanomata which commenced in the eyelids and conjunctiva had characters which did not differ from those in the skin generally, and they were amenable to the same treatment, which was surgical excision before they became malignant. Intra-ocular melanomata were beyond the control of surgery. Even removal of the affected eyeball did not prevent the patient ultimately dying of generalized cancer. He did not consider that either radium or X-Rays had been shown to be of any use in the treatment of these growths.

Dr. Arthur Whitfield, dermatologist at King's College Hospital, practically confined his attention to the discussion of **cellular nævi.** He considered that these were of two kinds: (1) papillomatous outgrowths, chiefly formed from the spinous layers of the epidermis; (2) those in which masses of cells were found lying in the true skin, really lobulations of new growth. The change was most marked, he said, at the base of the inter-papillary epithelial ridges, with the simultaneous solution of epithelial fibrils. In his view, the production of pigment was not an essential part of the nævus. There was, he believed, a solution of the prickles between the cells, hence they lost their structural continuity. Ordinary Paget's disease he regarded as malignant from the start. Often it was very difficult to say, when a melanotic growth had been removed and was submitted for an opinion, whether it was malignant or not.

Mr. E. Treacher Collins showed, by means of the epidiascope, a series of drawings of intra-ocular melanomata, both innocent and malignant. He gave his conclusions, which may be thus summarized. In the normal tissues of the eyeball, melanoblasts are both epiblastic and mesoblastic

in origin. In the iris, innocent melanotic growths may arise from both epiblastic and mesoblastic tissue. The innocent epiblastic melanomata may appear at the pupillary margin, and may be either cystic or solid. They may also extend into the stroma from its posterior surface. The innocent mesoblastic melanomata may be either localized or diffused. They are due to hyperplasia of the surface endothelium, or to the formation of clump cells, or to both combined. He said that clump cells are chromatophores in which the branching processes are retracted into the body of the cell, and where the melanin granules are concentrated.

Malignant growths may start from these innocent mesoblastic melanotic growths, and consist of spindle-shaped cells and clump cells. Metaplasia of endothelial cells into spindle-shaped cells takes place both in the normal development of the eye, after traumatic lesions, and in connection with malignant neoplasms. Innocent melanomata of mesoblastic origin, similar to those occurring in the iris, are met within the ciliary body and choroid. Malignant mesoblastic melanomata of the ciliary body and choroid may be classified in the following way: (1) Interfascicular endotheliomata, (2) peritheliomata, (3) sarcomata arising in the cells proper of the choroid. Malignant melanotic epithelial growths may, in rare instances, originate from the pigment epithelium of the ciliary body, and possibly also from the pigment epithelium of other parts of the uveal tract.

Mr. Affleck Greeves also discussed the subject, and showed a number of micro-photographs, some of which were taken by the late Mr. Coats. One case was that of a woman, aged 32, who had a pseudo-melanoma of the iris. She complained of mistiness of vision, and a dark brown spot was found at the periphery of the iris. Opposite the tumor the pupil was flattened. There was some keratitis punctate and some vitreous opacity. At a meeting at which the patient was shown, the general opinion was that it was a malignant growth, therefore the eye was excised. Subsequent examination, however, showed that it was inflammatory, there being only epithelioid and inflammatory cells. It was a gumma of the iris, and the Wassermann was positive. Three of her children gave a like reaction. Nævi of the conjunc-

tiva, when they became malignant behaved in the same way as carcinomata, and neighboring lymphatic gland became invaded.

Mr. Foster Moore spoke of innocent pigmented growths of the choroid. They were, he said, either congenital or developed quite early in life. As they did not show any change when watched for a number of years, he did not think they could be regarded as malignant.

Mr. Richardson Cross described a case of melanoma of the caruncle, in which the growth originated at the edge of the caruncle and plica. It lay along the surface of the palpebral conjunctiva as if it had been laid on with a paint-brush. The man did not complain of this; he had simply come for glasses. The growth had then been present four years, and the vision was not in any way impaired. A very radical operation was carried out, and the patient remained well thereafter 4½ years. At the end of this time there was a recurrence, and though there was no pigmentation and nothing could be seen in the eyeball, there was a gland at the angle of the jaw. Thorough evisceration of the eyeball was carried out. The specimen, which he exhibited, showed that the growth was melanotic sarcoma. It was well encapsuted.

The Value of Ultra-violet Light in Certain Eye Diseases.

Mr. Charles Goulden read a very interesting paper under this title, in which he said that ultra-violet light was a valuable means of curing tuberculosis of the iris and ciliary body. He gave details of a severe case in which, after several treatments had failed, ultra-violet light was used, with results which were miraculous. Phlyctenular disease was also greatly benefited by this means. He was not able to include episcleritis, sclerosing keratitis or chronic iridocyclitis in the conditions favorably influenced by these rays. In regard to their effect in central retinitis and sub-retinal exudation he suspended judgment.

Mr. W. S. Duke-Elder read a contribution on the same subject, and his views supported those expressed by Mr. Goulden.

Dr. W. O'Donovan spoke of the good effect of the rays in tuberculosis of the skin, especially lupus.

Siderosis Bulbi.

Mr. M. S. Mayou read a paper on this subject fortified by many good photographs and colored drawings.

Mr. R. Davenport read a contribution on **Senile Macular Exudative Retinitis.** None of the patients with this condition had retinitis proliferans. Blood pressure varied between 148 and 240. Most of the patients were free of albuminuria, but arterio-sclerosis was definitely present in some.

Miss Margaret Dobson read a paper on **Diseases of the Macular Region as seen by Red-free Light.** She contended that with this light, from a powerful source, many minute details in the examination of the eye were visible which could not be well discerned with the ordinary ophthalmoscope.

The subsequent speeches revealed some difference of opinion on the point.

Clinical Cases.

On Friday afternoon members attended the Ophthalmological Department of St. Bartholomew's Hospital, where Mr. R. Foster Moore had provided a considerable number of cases for examination. The discussion of them took place in the Library of that institution. The first were two cases of **Eale's Disease.** One of them was a woman aged 35, the other a man aged 25. Both had had spontaneous hemorrhages into the vitreous, and in both the Wassermann was negative. The blood coagulation-time was normal. No cause for the condition could be ascertained.

Growth in (?) the Chiasma.

This man came because of loss of sight. The upper half of the visual field in each eye had gone, and there was corresponding optic atrophy. X-Rays showed an apparently normal pituitary fossa, and the sugar-tolerance was normal.

At one time the man rapidly increased in weight, but he was now becoming normal again in that respect. He had had headaches, but there were no positive signs of a pituitary lesion. Mr. Moore suggested, for discussion, that the patient had a glioma of the chiasma. Thyroid had been administered, and the patient was now having pituitary extract.

Mr. J. H. Fisher said he considered that this was not a primary pituitary enlargement or tumor, but an extra-pituitary tumor. The symptoms were those of hypo-pituitarism, the pituitary body being probably deficient in function. The man had some lethargy and somnolence at one time, as well as adiposity, and the visual field also pointed to the condition being outside the sella turcica, implicating the pituitary body secondarily. If that were so, the administration of thyroid and pituitary would not be likely to benefit. He related the particulars of a case of his own with somewhat similar manifestations.

The President said he agreed the symptons in this case might be due to pressure on the third ventricle. It was possible, he thought, for glandular therapy to do good, because the view prevailing was that secretion from the posterior lobe of the pituitary got into the tissues via the cerebro-spinal fluid, and if there were a block in the secretion, the administration might act in a compensatory manner.

Rodent Ulcer Treated by Radium.

Mr. Foster Moore showed a patient from whom he removed a rodent ulcer from the inner angle of the eye a year ago, turning down a flap from the forehead to cover the operation area. She had been treated for some time with radium, and his experience of rodent ulcer treated by radium was unfortunate. Though the surface growth got smaller under the radium, the boring of the growth lower down still went on. In that way the ethmoid had become seriously implicated. Something further was now needed, and he asked what operation they considered was indicated. The pathological report was that the growth was rodent ulcer, not epithelioma.

Mr. Goulden said his experience of the use of radium in

these cases was similar to Mr. Foster Moore's. Often the aid of the ophthalmic surgeon was not sought until eye movements had become deficient, and it then usually meant that the orbit had become seriously invaded, also the nasal bone, the ethmoid, the floor of the frontal sinus, and the roof of the maxilla. In a recent case of the kind he operated in conjunction with the nasal surgeon, a course he recommended in these conditions.

MR. HARRISON BUTLER spoke of three cases of his own which had had radium applied, and which bore out what had just been said.

MR. AFFLECK GREEVES advised that the eye be sacrificed in this case, in the patient's interest.

Other cases were shown.

Birth Injuries of the Eye.

This was the subject of debate on Friday evening, when the obstetric physician's standpoint was presented by DR. COMYNS BERKELEY. He raised the question as to how many disabilities of the eye, regarded as congenital, could be truly said to be due to injuries inflicted on it during the process of birth. After thirty years of busy obstetric practice his view was that injuries of the eyes of infants caused during birth were of very rare occurrence. But it was not his practice to examine the eyes of infants with the ophthalmoscope. Incidentally, he declared himself as averse to the use of instruments at labor if their employment could be done without.

A full discussion followed.

Optic Neuritis in a Pedigree Bull.

MR. A. F. MACCALLAN and MISS IDA MANN read papers setting out their findings in the case of a pedigree bull in which optic neuritis resulted in blindness, so that the animal had to be destroyed. Owing to some delay, the brain was not preserved, and therefore could not be reported upon. The first paper was concerned with the ophthalmoscopic observations during life, the second with the histology and

pathology. The animal's sight had been good until eight months previously. When seen at the Royal Veterinary College both pupils were dilated and immobile. The only abnormality visible was a small hemorrhage. There appeared to be only bare perception of light. Miss Mann found that the disk was swollen, and that there was a large round-celled infiltration. The swelling was due to œdema. The physiological cup was filled up, and the central vessels were widely dilated. Far back, the optic nerve was normal.

The Fincham Slit-lamp.

Mr. Fincham described and demonstrated his slit-lamp, and also showed by means of the epidiascope some remarkably fine microphotographs of the human eye.

On Thursday evening the Congress Dinner took place, the President being in the chair; and during the days of the Congress a small but very varied collection of instruments was shown in an adjacent room.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

The Section held its usual meeting on the 14th of May, Sir Arnold Lawson, K. B. E., president, in the chair.

Mr. Rayner D. Batten demonstrated a large series of pictures, illustrating (1) Black-spot choroiditis, (2) Senile retino-choroidal atrophy of the macula.

Black-Spot Choroiditis

He said the black-spot choroiditis was met with in people who had high myopia, but it was not limited to myopia, as it was found also in hypermetropia. The central area was the part affected. He recognized three forms of black-spot choroiditis. In the first of these the onset was acute, the whole process occurring with dramatic suddenness, and the area surrounding the black-spot quickly became atrophied. In his experience, hemorrhage had never been a marked feature of the condition. In the second class the onset was less acute, and later on the choroidal atrophy was liable to become less. The eye might regain a useful degree of vision.

Senile Retino-choroidal Atrophy of the Macula.

This Mr. Batten regards as a definite class of macular disease. The first few cases of the kind which he saw gave evidence of cerebral disturbance, and he formed the idea, in consequence, that as there was a real association between macular disease and cerebral degeneration it was only reason-

able to expect the same in the adult of fairly advanced age, and still more so in the old. In later cases, however, there did not seem to be any cerebral degeneration, but physicians declared that the patients had arteriosclerosis, and that otherwise they enjoyed good health for their age. The ocular condition he was describing occurred in both old and young, was of long duration and slow formation, and was remarkably symmetrical in the two eyes. It occupied the macular area, and was of discoid shape. The superficial layers of the choroid were lacking, leaving choroidal vessels coursing across the base. At the margins there were retinal changes, and the kind of fine pigment loss which he took to be evidence of retinal disease. Vision was very bad in most of the cases. The symmetry bespoke constitutional disease.

THE PRESIDENT said three of the patients whose condition was illustrated by Mr. Batten were his own, and in certainly two of them the teeth were very bad. On the mouth being attended to the development of the eye disease was stayed. He regarded the presence of pigment as denoting inflammation rather than hemorrhage.

MR. J. H. FISHER considered that the accumulation of pigment was derived from the pigment layer of the retina, and that the disorder of pigmentation was probably a result of imperfect nutrition of that layer by the chorio-capillaris. In the sclerotic cases there was atrophy of the chorio-capillaris, conspicuous over the central circular area with an accumulation of pigment at the periphery, and little or no pigment where the chorio-capillaris was deficient over most of the area. With regard to the difference in the two classes of cases, it seemed possible that where, in the arteriosclerotic cases, there was obstruction of a branch of an artery conveying blood to the chorio-capillaris, the blood was cut off except at the periphery, and at the periphery it was at a low ebb. In regard to the myopic cases, there was a tendency in these for fissures to occur in the choroid coat, and if these fissures formed a circular crack, this was favorable to choroidal rupture.

MR. E. TREACHER COLLINS said he thought the general view was that the black spots at the macula were due to hemorrhage; indeed he had watched a hemorrhage gradually

become a black spot. The blood came from the choroidal vessels, in his view, and as the onset was sudden, hemorrhage seemed to be the most likely explanation. Though these patients suffered a sudden loss of central vision, one could assure them they would still retain a degree of sight.

MR. H. R. JEREMY referred to the case of a boy under his own care who, at 17 years of age, was blind in one eye and suddenly lost his sight in the other while at a dance. He had high myopia. In one eye was a black spot at the macula, in the other a large hemorrhage below the macula. The boy was put to bed six weeks, and at the end of that time he had sufficient vision to enable him to get about.

MR. J. F. CUNNINGHAM related the case of a lady who, in 1910 had 6/9 vision, and myopia–10. Somewhat rapidly she developed very black spots, and in one eye she had a circular detachment. Since the war, in place of the dark spots she had developed a large area of choroidal atrophy, with sclerosis of vessels.

REPORT OF THE PROCEEDINGS OF THE SECTION OF OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By THOMAS H. CURTIN, Secretary.

MEETING OF MARCH 15TH, 1926. CLYDE E. MCDANNALD, CHAIRMAN.

Dr. A. Milton Goldman (by invitation) read the first paper of the evening entitled, **a stereoscope for tangent screen examination.** Two prisms (10 P.D. each) are placed base out in a wooden frame, 15*cm* x 26*cm*. The lenses are round and measure 47*mm* in diameter, thus providing sufficient peripheral field. The pupillary distance is 6½*cm*. The nasal screen is 8*cm* wide and 22½*cm* long and made of light metal. An adjustable chin rest is used. The entire stereoscope is painted dull black. It is placed on a tripod having a heavy base so as not to be easily moved by the patient during the examination. The instrument is to be used at a distance of ½ meter from the fixation points and the one meter tangent screen devised by Dr. A. H. Thomasson has been used since it simplifies the charting of the fields. The radii of the tangent screen circles for binocular fixation at ½ meter are then ½ the radii for monocular testing with Thomasson's one meter tangent screen; thus the same record chart may be used. Two white headed pins (3*mm* in diameter) 10*mm* apart are used as fixation points against the black surface of the screen. One pin is in the center of the screen and corresponds to the eye tested. To this stereoscope Dr. David Wells of Boston has very kindly devised a quick preliminary test chart for detecting any muscle imbalance which might interfere with fusion, so if necessary, prisms taken from a

trial case may be added to procure orthophoria. Patient should wear correction lenses during test. With this simple and inexpensive stereoscope fusion is readily obtained. There is practically no chromatic aberration and the patient does not tire easily. The Blind spots of Mariotte at this distance, (½ meter), measure 5*cm* x 7½*cm* and the peripheral fields are wide enough to include all central and paracentral defects. The main advantage, however, is obviously one of obtaining greater magnification of the blind spots and central fields than by any other binocular test. The instrument is easy to construct.

The second paper of the evening was read by DR. A. H. THOMASSON, (by invitation). **A simplified scotometer with suggestions for field taking.** This paper will be printed in full in the ARCHIVES OF OPHTHALMOLOGY. His paper describes a new tangent screen. The paper was illustrated by stereoscopic views.

DISCUSSION: DR. DUANE thought Dr. Thomasson's rigid frame a very good point.

DR. TRONCOSO in his discussion stated that Dr. Thomasson is using the anatomical method of registering the visual field, proposed by him in this section more than one year ago. He discussed the difference between the two methods. He wished also to emphasize the importance of the use of the screen in all cases of subacute and especially simple glaucoma, saying that the beautiful slides of Dr. Thomasson showed very clearly the arcuate scotoma and several paracentral defects in these conditions. With a simpler and less expensive device such as this, the Bjerrum screen will probably be more extensively used to the great advantage of the patients. The progress of the scotoma and the findings with the genioscope give excellent date for the prognosis of glaucoma, the necessity of an operation, and its results.

DR. J. N. EVANS (by invitation) presented a contribution on the **scotoma caused by the retinal blood vessels.** A review of the literature showed the previous attempts to map the vessels had been unsuccessful. Many slides were shown of what Dr. Evans stated were vessel scotomata taken on a short radius campimeter with a ½° white object. Most of these slides were from normal fundi, and showed numerous

vessels, even to the smallest branches visible by means of the ophthalmoscope.

DISCUSSION: DR. RALPH I. LLOYD said that he felt a good many cases of glaucoma are associated with vessel scotoma.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lancton Foster, M.D., F.A.C.S., New Rochelle, N. Y.

(*Continued from July issue.*)

XIII. MISCELLANEOUS.

317. Becker. **A case of aniline pencil poisoning of the eye.** *Muench. medizinische Wochenschrift*, 1922, p. 290.

318. Black, N. M. **Siderosis with dilated inactive pupil. Recovery.** *American Journal of Ophthalmology*, December, 1923.

319. Bourgeois. **Apropos d'une blessure oculaire par plume d'ecolier.** *Archiv d'Ophtalmologie*, xxxix., 1922, No. 2.

320. Elliot. **Memory sight.** *British Medical Journal*, February, 1924.

321. Finnoff, William C. **Changes in eyes of rabbits following injection of dead bacilli into common carotid artery.** *American Journal of Ophthalmology*, May, 1924, p. 365.

322. Francis, Lee Masten. **Adenocarcinoma probably arising from lacrimal gland.** *Ibid.*, p. 260.

323. Lampert. **De la tolerance du crytellin pour les petits Corps etrangers metalliques. La difficulte du diagnostic.** *Arch d'Ophtalmologie*, December, 1921, p. 745.

324. McKee, S. H. **War lesions of the fundus.** *American Journal of Ophthalmology*, September, 1923.

325. Metzger. **A case of nearly complete iridodialysis.** *Klin. Monatsbl. f. Augenheilkunde*, January–February, 1922, p. 245.

326. Villard. **Deux cas de myase oculaire.** *Annales d'oculistique*, clix, No. 9, p. 670.

327. Wright, R. E. and Naidu, T. V. **Eclipse amblyopia.** *American Journal of Ophthalmology*, October, 1923.

Becker (317, **Aniline pencil poisoning of the eye**) saw a case of this nature and placed it under treatment about seven-

teen hours after the injury. The methyl violet had already caused a diffuse opacity of the cornea and a large necrotic burn of the conjunctiva. A mixed injection and great photophobia persisted for a long time after the stain had disappeared. The prognosis for such moderately severe cases seems to be good. The importance of early, frequent, and thorough irrigation is emphasized.

BOURGEOIS (319, **Wound of the eye by a schoolboy's pen**) describes the condition as a black line running in the vertical meridian of the cornea, ending above and below one millimeter from the limbus. He believes this to have been due to the vertical position of the pen with reference to the eye and the protective movement of the latter. To remove the ink he used javelle water applied with a fine pencil after cocainization of the eye. The upper two-thirds of the tattooing had disappeared by the following day and after waiting another day the treatment was repeated. Two weeks later all that could be seen of the corneal injury was a paracentral minute white spot below. The vision was 0.8.

Lieutenant-Colonel ELLIOT (320, **Memory sight**) says that the visual sensations of persons who have become totally blind is a matter of interest to the ophthalmic surgeon who is supposed to know all about these things by the laity. According to Elliot it is characteristic for such persons to see a "bright background" which they describe variously as "a bright cloud" or as "a luminous haze" or as "a play of colours." The sensation of blackness is certainly as positive a sensation as any colour and there is no more reason why the totally blind should live in perpetual black darkness as is popularly supposed to be the case than in any other colored surroundings and further if this was so it would be likely to have a depressing effect upon blind persons whereas as Elliot points out they are remarkable for their cheeriness. Elliot in this article speaks of another feature of the "sight of the blind." This feature he speaks of as "memory sight." This is a condition in which the blind person states that he sees objects about him and even can picture scenes passing around him. This of course is a psychological phenomenon and is one which is very much valued by blind persons. Elliot speaks of a case of an elderly patient who was absolutely blind in

each eye from glaucoma, and in whom it was necessary to remove one eye which had become painful. This patient was much disturbed as to whether the removal of the eye would deprive her of her subjective sensations of sight which caused her so much pleasure. The enucleation of the eye made no difference to these sensations.

P. G. DOYNE.

BLACK (318, **siderosis with dilated inactive pupil**) reports a case of siderosis in an eye which harbored a piece of steel for six months. The pupil was dilated and immobile. After the extraction of the foreign body the pupil became equal to the other and reacted slightly. The siderosis partly disappeared. The author asks the following questions: Why do some eyes with retained steel escape discoloration? Why does the pupil sometimes show mydriasis and immobility? Why in some eyes are there deposits beneath the lens capsule with or without lens changes? The observations of Verhoeff which demonstrated the presence of oxide of iron in the sphincter and dilator muscles may have some bearing on these problems. Also the fact that steel particles vary in chemical composition may be the cause of the absence of discoloration in some cases. ALLING.

In both the cases reported by LAMPERT (323, **Tolerance of the lens to small metallic foreign bodies**) minute bits of iron were located in the lens by means of the corneal microscope and slit-lamp. The lenses remained clear, the wounds in the capsule were closed in one case with iris pigment in the other by a clot of fibrin.

FINNOFF (321, **Changes in eyes of rabbits following injection of dead bacilli into common carotid artery**) produced ocular tuberculosis in from two days to nine weeks in rabbits by injecting dead bacilli in the common carotid artery. The lesions resembled those which he produced in animals by injecting living bacilli. The essential difference is in the progress of the lesion and the duration of the animal's life. Living bacilli produce severe and progressive lesions in the eye in addition to pulmonary tuberculosis from which the animals die before the eye lesions have an opportunity to run the full course. With dead bacilli, ocular lesions were observed during life and examined histologically afterwards

in the iris, cornea, conjunctiva, lids, choroid, retina, vitreous and optic nerve. ALLING.

FRANCIS (322, **Adenocarcinoma probably arising from lacrimal gland**) reports a case in which malignancy was not recognized until patient sought restoration of the socket. From the pathological examination it was difficult to ascertain, whether the tumor was a remnant of the old embryonal growth for which the eye was enucleated in the early life of the patient or a new growth originating from remnants of lacrimal duct. ALLING.

MCKEE (324, **War lesions of the fundus**) illustrates twenty-eight cases of ocular lesions with drawings and diagrams which indicate the character and situation of the injuries. They are mostly cases of choroiditis, retinochoroiditis, rupture of the choroid and macular changes. Some of the injuries were produced by "windage" or air commotion due to nearby explosion. ALLING.

After a contusion of the cornea METZGER (325, **Nearly complete iridodialysis**) saw the iris torn away throughout five-sixths of its periphery. The lens with its attachments remained intact, and no injuries could be found in the fundus.

VILLARD (326, **Two cases of ocular myiasis**) reports two cases in which severe pain set in after the sting of an insect near the eye. In the conjunctival sac of each were found numerous, very small, movable foreign bodies, which proved under the microscope to be the larvæ of flies. Rapid recovery followed washing them out.

WRIGHT and NAIDU (327, **Eclipse amblyopia**) record twenty-one cases of injuries which occurred during the eclipse of the sun last year. All were mild and showed a marked and rapid improvement. They suggest that the deeply pigmented eyes of the Indian may have protected the eyes from more serious damage which often befalls patients of other countries. ALLING.

(*Concluded.*)

I.—THE EYE IN ITS RELATIONS TO GENERAL DISEASE.

1. ABELSDORFF. **Another case of optochin amblyopia with patches of chorioretinal degeneration.** *Deutsche med. Wochenschrift*, 1923, p. 792.

2. ADIE. **Cataract in dystrophia myotonica (myotonia atrophica).**

3. ALAJMO. **Parenchymatous keratitis in the course of a case of glomerular nephritis.** *Bolletino d'oculistica*, January, 1923.

4. BAILEY, P. **The function of the hypophysis cerebri.** *Ergebnisse der Physiologie*, 1922, xx., p. 162.

5. BROWN, ALBERT L. **Ocular manifestations in serum sickness.** *American Journal of Ophthalmology*, August, 1925, p. 614.

6. BUSACCA. **Clinical contribution to the ocular lesions met with in lethargic encephalitis.** *Bolletino d'oculistica*, 1923, No. 9.

7. CHENEY, R. C. **The toxemias of pregnancy from an ophthalmological standpoint.** *Journal A.M.A.*, lxxxiii., p. 1383.

8. CUMMINS. **Hyperplasia of the hypophysis cerebri,** *British Journal of Ophthalmology*, February, 1923.

9. DUKE-ELDER, STEWART. **Changes in refraction in diabetes mellitus.** *Ibid.*, April, 1925.

10. GAUDISSART. **The phantasies of hysterical amblyopia.** *Annales d'oculistique*, clx., May, p. 380.

11. GONZALES, J. DE J. **Pathogenesis of intrinsic ocular paralysis during convalescence from acute infectious disease.** *American Journal of Ophthalmology*, May, 1924, p. 351.

12. HAISST. **Ocular symptoms in lethargic encephalitis.** *Vereinigung Wuerttemberger Augenaerzte at Tuebingen*, June 10, 1923.

13. V. HORVATH, BELA. **Formation of cataract during influenza.** *Klin. Monatsbl. f. Augenheilkunde*, 1923, lxx., p. 521.

14. LANGDON, H. M. **Ocular complications of chorea.** *American Journal of Ophthalmology*, August, 1925, p. 626.

15. LEE, T. P. **Ocular findings in Kala-Azar in Central China.** *American Journal of Ophthalmology*, 1924, p. 835.

16. LILLIE, WALTER I. **Ocular phenomena in acromegaly.** *Ibid.*, January, 1925, p. 32.

17. LINK. **Epidemic mumps.** *Archiv f. Ohren-, Nasen- und Kehlkopfheilkunde*, iii., 1, p. 1.

18. MILLS, LLOYD. **The significance of ocular changes occurring in association with pre-eclamptic symptoms.** *American Journal of Obstetrics and Gynecology*, 1924, 3, p. 304.

19. MORAX and LAGRANGE. **Bilateral choked disk due to tumor of the aqueduct of Sylvius. Extradural hemorrhage after lumbar puncture.** *Annales d'oculistique*, clx., 6, p. 445.

20. RIDDOCK and GOULDEN. **On the relationship between subarachnoid and intraocular hemorrhage.** *Proceedings of the Royal Society of Medicine.*

21. SATTLER, C. H. **Bromural and adalin poisoning of the eye.** *Klin. Monatsbl. f. Augenheilkunde*, 1923, p. 149.

22. SCHUBENS, H. **Bilateral paralysis of the abducens and choked disk after lumbar anesthesia with novocain-suprarenin.** *Ibid.*, 1923, p. 154.

23. SEGI, M. **Retinal hemorrhages in purpura hemorrhagica.** *Ibid.*, lxxi., p. 53.

24. TWELMEYER, O. **Metastatic ophthalmia.** *Vereinigung d'Augenaerzte Schlesiens und Posens*, Breslau, March 4, 1923.

25. WALSHE. **A case of secondary carcinomatous infiltration of the pia-arachnoid of the brain, presenting exclusively ocular symptoms during life: Meningitis carcinomatosa.** *British Journal of Ophthalmology*, January, 1923.

26. ZENTMAYER, WILLIAM. **Obstruction to central artery of retina during an intranasal operation.** *American Journal of Ophthalmology*, March, 1925, p. 211.

ABELSDORFF (1, **Optochin amblyopia with patches of chorioretinal degeneration**) reports the case of a woman 35 years old who in 1914 took three doses of 0.25*gm* of optochin on account of pneumonia and was blinded by it. At the time of writing the vision of her right eye was $\frac{6}{20}$, that of her left $\frac{6}{36}-\frac{6}{25}$. She has marked hemeralopia, great contraction of the visual field, and a paracentral scotoma for all colors. In addition to the fundus changes commonly found in optochin amblyopia there are several whitish depigmented spots with black masses of pigment in the region of the macula. The choroidal vessels are visible in the depigmented places and have sclerotic walls. The author thinks that the optochin produced not only changes in the retina, but also a thickening of the walls of the choroidal vessels, and that the sclerosis of the choroid resulted in a chorioretinal degeneration with pigmentation, such as is met with in sclerosis of the choroid without intoxication.

SATTLER (21, **Bromural and adalin poisoning**) reports two cases. In the first a man 42 years old had taken twenty tablets of bromural daily for 11 years, in the second a man 49 years old had taken from six to ten tablets of adalin daily for 10 years. In each case there was a gradually increasing impairment of vision with a scotoma extending from the blind spot to the point of fixation and an atrophy of the papillomacular bundle of optic nerve fibers. Aside from a reflex immobility of the pupils in case 2 there were no other neurological symptoms. Considerable improvement followed stoppage of the drugs.

ADIE (2, **Cataract in dystrophia myotonica (myotonia atrophica)**) draws attention to the importance of cataract as a feature in this disease. Myotonia atrophica is an heredofamilial condition characterized by atrophy of the muscles of the face and of the sternomastoids, the vasti and peronei muscles, myotonia, most conspicuous in the hand grasps,

and extra-muscular symptoms especially cataract, atrophy of the testicles with impotence, baldness and vasomotor disturbances. The members of earlier generations do not usually suffer from muscular symptoms but are very prone to develop cataract at a rather earlier age—presenile cataract. It is probable that when this is more widely realized many more cases will be recognized. The fully developed condition seems to be confined to one generation but the disease appears to afford an example of "anticipation" and of the progressive deterioration of an affected stock. The actual form of the cataract does not appear to show any very characteristic features though in a fair proportion of those cases in which detailed notes of the lens changes were made it was stated that there was a gray star shaped figure at the posterior pole of the lens.

P. G. DOYNE.

ALAJMO (3, **Parenchymatous keratitis in a case of glomerular nephritis**) met with a case of this nature in a girl 18 years old. The disease was confined to the right eye and began without any noticeable inflammatory symptoms or subjective troubles with a semicircular superficial opacity in the parenchyma of the lower margin of the cornea. In a few days the opacity had extended to the middle of the cornea, still confined to the superficial layers of the parenchyma, and at the same time a typical gerontoxon formed in the lower part of the limbus. In this stage the vision was much impaired, there was a slight hyperæmia of the iris, and some conjunctival irritation. Clinical investigation revealed a mild glomerular nephritis, with no signs of tubercular or syphilitic disease. Simultaneously with the disappearance of the albumin from the urine on the sixteenth day the corneal opacity cleared up except for a vascularization which persisted a few days longer and then passed away leaving no trace of the disease. At the end of two months the patient had regained perfect vision.

BAILEY (4, **Function of the hypophysis cerebri**) says that the histological structure of the anterior part of the hypophysis is glandular, that its secretion has not yet been isolated, and that the effect which it produces upon the organism has not yet been established. The posterior part is not glandular, its removal from the body induces no symptoms, but from it a

substance can be isolated which is very active when injected into animals. The chemical structure of this substance is unknown. It has not been proved that this substance is the product of secretion from the posterior part, and there is evidence to the contrary.

CUMMINS (8, **Hyperplasia of the hypophysis cerebri?**) reports the case of an unmarried woman 33 years old, who had been under gynecological treatment for a year because of amenorrhea, commencing adiposity of the genitals and general weakness. Her uterus was somewhat infantile. In January, 1922, the vision of her right eye was $\frac{1}{60}$, that of her left $\frac{6}{18}$. She had bitemporal hemianopsia and hyperæmia of the papillæ. The X-rays showed no pathological condition in the region of the sella turcica. These symptoms taken together with her general condition led the author to suspect a hyperplasia of the hypophysis with a consequent impairment of the function of the thyroid. The results obtained by a prolonged administration of thyroidin confirmed him in this opinion. The vision improved in each eye to $\frac{6}{6}$ within six months, the fields became normal and the general condition good.

From a study of seventy-five cases, BROWN (5, **Ocular manifestations in serum sickness**) finds vascular manifestations which he attributes to dilation of the blood vessels. The disk vessels become prominent first, giving a pinkish tinge to the optic nerve. The disk margin becomes hazy particularly on the nasal side, accompanied by noticeable œdema in severe cases. ALLING.

BUSACCA (6, **Ocular lesions met with in lethargic encephalitis**) reports the observations made in five cases of encephalitis. In two there was transient diplopia; in two bilateral, in one monolateral ptosis; in three paralysis of the abducens; in two bilateral, in three monolateral paralysis of the convergence; in four paralysis of the accommodation; in four monolateral spastic miosis; in five slow pupillary reaction; in three hypæsthesia of the cornea; in three contraction of the visual field, and in two enlargement of the blind spot. All the patients recovered.

HAISST (12, **Ocular symptoms in lethargic encephalitis**) likewise reports on five cases. Three had somnolence with ptosis. In all five diplopia appeared as an early symptom.

Three had paralysis of the abducens; three disturbance of the accommodation. Symptoms that persisted during convalescence were in one case anisocoria and slow pupillary reaction; in one disturbance of the accommodation, and in a very severe case absolute immobility of one pupil. Disturbances of the accommodation lasted longer than the paralysis of the muscles. In another case, which was probably one of lethargic encephalitis, in which at any rate syphilis had been excluded, there was a true reflex immobility of the pupils.

CHENEY (7, **The toxemias of pregnancy from an ophthalmological standpoint**) reviews the literature on the subject and also his experience in this field. He concludes that fundus changes are most common in the latter three months of pregnancy. It seems that most of the cases in which retinitis occurs are of two types: (1) acute toxemia and (2) chronic nephritis. In the former, retinitis is found less frequently. The ocular disturbance is practically always acute even in patients with chronic nephritis, in whom there is really an acute flare-up of what is usually a low grade chronic process. Granting all the possibilities of the various theories as to the genesis of nephritic and toxemic retinitis, he feels that Volhard's theory of retinal ischemia is the best for the present, although it is far from being entirely satisfactory.

ALLING.

MILLS (18, **The significance of ocular changes occurring in association with pre-eclamptic symptoms**) finds that 90% of all cases of pregnancy show some form of eye envolvement as a result probably of the physiological enlargement of the pituitary gland. The pressure of the gland on the chiasm may cause different degrees of contraction of visual fields, and in some cases a temporary but decided loss of central as well as temporal vision have also been noted. A retinal stasis and œdema are occasionally seen without any evidence of gross renal, blood or obstetric pathology.

ALLING.

GONZALES (11, **Pathogenesis of intrinsic ocular paralysis during convalescence from acute infectious disease**) believes that substances such as the toxins of Ehrlich's side chain theory arise out of the action of the bacterial toxin and the body cells which are reacting against the toxic attack. Dur-

ing convalescence the product may be present in the blood and paralyze the neuromuscular synapses temporarily. It is not a specific, inasmuch as various toxins cause its production in unrelated cases of diphtheria, typhus, pneumonia and influenza. ALLING.

Sudden changes in the refraction of the eye are a well known feature in the ocular complications of diabetes mellitus. The change most commonly noticed is one towards myopia: only occasionally is a change towards hypermetropia noticed. DUKE-ELDER (9, **Changes in refraction in diabetes mellitus**) suggests that these changes may be directly correlated with the blood sugar content. He describes in detail three cases. In the first the refraction varied directly with the blood sugar content, in the second hypermetropia developed following a decrease of sugar, and in the third myopia developed as a terminal complication of fatal diabetes. It would appear that there is a tendency to hypermetropia with decreased sugar and to myopia with increased sugar. With the advent of insulin, the proportion of hypermetropia-developing cases will probably increase.

Duke-Elder discusses the possible and probable causes of these refractive changes and concludes that, except in a few cases, where paresis of the accommodation in part accounts for the change to hypermetropia, the phenomenon is due to osmotic processes involving the lens, caused by a variation of the molecular concentration of the blood and tissue fluids with the sugar content; that the tendency is to return to normal, and that, provided that the diabetic element can be successfully dealt with, an invariably good prognosis as regards vision can be given. P. G. DOYNE.

LANGDON (14, **Ocular complications of chorea**) reviewing the literature finds that organic ocular changes occurring during attacks of chorea are rare. The more important complications are obstruction of the central retinal artery or its branches followed by optic atrophy, and sometimes changes in the ocular motor nuclei or nerves resulting in weakness of the muscles and diplopia. ALLING.

GAUDISSART (10, **The phantasies of hysterical amblyopia**) reports two cases of hysterical amblyopia which presented peculiar symptoms. The first patient was a woman 29 years

old who had rapidly lost her vision within a week. She complained of intense photophobia; her pupils reacted rather slowly; her visual fields for white were irregularly concentrically contracted, and for colors to at most ten degrees. The conjunctival reflex was absent. In all other respects the eyes appeared to be normal, and a general examination revealed nothing abnormal elsewhere. After a few months her vision again became normal. Noteworthy points are that considerably less could be read with each eye separately than with the two combined, that in spite of emmetropia the vision was brought up to normal for distance with − 2.5 D spherical, while no near vision could be obtained either without a glass, or with + 3D spherical. After a few months more all pathological symptoms had disappeared. The second patient, a woman 22 years old, had noticed impairment of her sight for two months. She also had photophobia with much lacrimation. The visual field was as in the first case, with a central scotoma for colors. The course followed was similar to that in the first case.

v. HORVATH (13, **Formation of cataract during influenza**) says that at the end of the second week of the disease a partial cortical cataract was found in the right eye which reduced the vision to $\frac{5}{10}$. The lens capsule presented a shagreened appearance. Just beneath this in the cortex were irregular and dense opacities, some radiating, some arching backwards from the superficial layers into the deeper ones. In the center was an irregularly quadrangular, whitish, lustrous opacity. The opacities were, except for the central, formed of smaller and larger vacuoles. The posterior embryonal suture of the lens nucleus was quite visible with a chalky white, punctate opacity about it. Remains of the hyaloid could be seen. The author tries to differentiate the opacity as described from a congenital opacity of the lens, and thinks that in its production the action of the toxine of influenza may have played a part.

LEE (15, **Ocular findings in Kala-Azar in Central China**) found multiple retinal hemorrhages in four cases out of 140 which he studied. Two patients also had subconjunctival hemorrhages. He thinks the latter probably due to the treatment with potassium antimony tartrate. The retinal hemorrhages were found close to the disk and along the blood vessels.

ALLING.

LILLIE (16, **Ocular phenomena in acromegaly**) summarizes the ocular condition found in fifty cases, sixty-eight per cent. of all patients with acromegaly in this series were normal ophthalmologically so far as any chiasmal changes of the optic nerve were concerned. Sixteen per cent. had definite ocular changes characteristic of chiasmal involvement in which the sella turcica was enlarged with erosion of the posterior clinoids.

ALLING.

LINK (17, **Epidemic mumps**) observed the following ocular complications during an epidemic of mumps: Optic neuritis and subsequent optic atrophy; disturbances of the accommodation; iridocyclitis; opacities in the vitreous and retina, and exophthalmos. The optic neuritis was due to inflammation at the base of the brain. The other pathological conditions are ascribed to the toxic influence of the mumps infection.

ALLING.

RIDDOCK (20, **On the relationship between subarachnoid and intraocular hemorrhage**), dealing with the neurological aspect, said that ætiologically there were 3 main groups of cases:—

(1) Traumatic rupture of meningeal vessels.

(2) Primary intracerebral hemorrhage.

(3) Nontraumatic rupture of a meningeal vessel or aneurysm.

With regard to symptoms; previous to the actual seizure certain symptoms may be elicited, and, of these, headache, giddiness, defects of memory for recent events and tinnitus are the most usual. The onset of the seizure is sudden and is usually associated with some straining or effort. There may be almost immediate death, but more characteristic is a clinical state suggestive of meningitis—after the early phase of coma, a state ensues in which there is clouding of consciousness, disorientation and delirium. Questions may be answered more or less sensibly, but there is usually much resentment of interference. One or other of the cranial nerves is usually affected. Abdominal reflexes are diminished, tendon jerks are increased, and the plantar response is extensor. Kernig's sign is often positive, and there is usually rigidity of the neck. A most important and invariable sign is the presence of blood in the cerebro-spinal fluid. Recovery

from the first seizure is common, and some patients have survived several.

GOULDEN described in detail the anatomical relations of the optic nerve and its coverings, and showed that both the subdural and the arachnoid space ended in a cul de sac on reaching the globe, and that no communication between the subarachnoid space and the interior of the globe had ever been demonstrated. With regard to the ophthalmoscopic signs in cases of hemorrhage into the nerve sheath, papillœdema, bilateral and of moderate amount, is nearly constantly present. It is of rapid onset. The retinal veins are always engorged and tortuous. Retinal hemorrhages, similar to those seen in other conditions, are common. Pre-retinal and vitreous hemorrhages are often seen. He described the pathological examination of an eye from a fatal case of subarachnoid hemorrhage in which there were retinal hemorrhages, and in which the subarachnoid sheath of the nerve was distended with blood. But yet careful serial sections could demonstrate no communication between the subarachnoid sheath and the interior of the eye. He came to the conclusion that the retinal hemorrhages were produced by pressure upon the veins by the distended subarachnoid space, and in consequence by interference in the venous return from the eye.

PATON in the discussion, said that cases were met with in which there were large subhyaloid or pre-retinal hemorrhages with no distension of the veins and no sign of papillœdema. He thought it was difficult, in these cases, not to suppose that there must be some direct communication between the subarachnoid space of the nerve and the interior of the eye.

P. G. DOYNE.

MORAX and LAGRANGE (19, **Bilateral choked disk due to tumor of the aqueduct of Sylvius**) report a case of this nature in a woman 34 years old who died as the result of an extradural hemorrhage following lumbar puncture. Five *ccm* of fluid were withdrawn, which showed nothing pathological except a slight increase of pressure and a little increase of cells. Autopsy revealed a hæmatoma as large as a hand over the left frontal lobe, and a rose colored tumor which filled the dilated aqueduct of Sylvius. It extended from the level of the

anterior half of the lamina quadrigemina down into the fourth ventricle. The lateral and posterior horns of the ventricle were much distended. Under the microscope the tumor proved to be a vascular neurocytoma of epithelial nature.

SCHUBENS (22, **Bilateral paralysis of the abducens and choked disk after lumbar anesthesia with novocain-suprarenin**) describes the findings in an 18-year-old girl some days after the operation. In addition to the troubles mentioned in the title there was anisocoria and small retinal hemorrhages near the left papilla. Eight months later complete recovery had taken place, except for a persisting anisocoria. Schubens thinks that an irritation of the meninges was produced which led to an increased secretion of fluid, and calls this condition toxic serous meningitis. By this assumption he is able to explain all the symptoms, such as vomiting, vertigo, and severe headache, as well as the ocular symptoms. The paralysis of the abducens is taken to be due to pressure. The prognosis in such cases is good. The only therapeutic measure recommended is repeated lumbar punctures.

SEGI (23, **Retinal hemorrhages in purpura hemorrhagica**) observed a hemorrhagic retinitis with marked involvement of the optic nerve in a case which presented the general features of purpura hemorrhagica. Aside from these changes the eyes were perfectly normal. The vision of each eye was fingers at five feet. In each fundus there was a superficial hemorrhage starting from the fovea centralis and sinking downward. In addition a large number of extravasations, some roundish, others irregular, formed almost a circle about the papilla. Neuritis was present with great venous engorgement. The microscope showed a flat detachment of the retina. An albuminous exudate in the subretinal space with a fibrous network in places, some cells, and also red blood corpuscles. The retina was much swollen. Mueller's supporting fibers stood out very clearly. The nerve fiber layer was much broadened. The granular layers showed a marked irregularity in the thickness of its layers, that of the rods and cones being well preserved. Hemorrhages were present in all layers, the most extensive ones in the layer of nerve fibers and in the outer reticular layer. Pigment was scattered about freely in the layer of nerve fibers. The changes grew less toward

the periphery, yet were quite marked in the ora serrata. The head of the optic nerve showed œdema and increase of cells. The vitreous and aqueous were rich in albumin, the vitreous contained many round cells. The iris was infiltrated with round cells.

TWELMEYER (24, **Metastatic ophthalmia**) reports four cases, in three of which the occurrence led to the diagnosis of the general disease. The fundamental disease was measles in one case, meningitis in the others, in one of which the meningococcus intracellularis was found by lumbar puncture.

ZENTMAYER (26, **Obstruction to central artery of retina during an intranasal operation**) cites a case which is quite similar to some previously reported. The route through which an embolus might pass from the nose to the central retinal artery is discussed. ALLING.

WALSHE (25, **Secondary carcinomatous infiltration of the pia-arachnoid of the brain presenting exclusively ocular symptoms during life**) reports the case of a widow 57 years old who had suffered for six months from headache, with an increasing paresis of the left abducens, temporal pallor of the papilla, and reflex immobility of the pupil. Vision sank to movements of the hand at about half a meter, while the field remained normal. Wassermann negative. Five weeks after entrance into the hospital she died, the end being preceded by a rise of temperature and psychomotor disturbances. Autopsy revealed an old tuberculosis at the left apex, an old mitral stenosis, and a bronchopneumonia of both lower lobes. Macroscopically the brain did not show much except a slightly thickened left abducens. But the microscope showed that the pia and arachnoid were greatly infiltrated with carcinomatous cells. These had also penetrated the substance of the pons to its ventral surface, had involved the walls of the blood vessels, and had infiltrated the left abducens and optic nerve. The primary tumor was not found, but from the appearance of the carcinomatous cells its location was suspected to be somewhere in the gastrointestinal tract.

(*To be continued.*)

NOTICE.

American Board of Otolaryngology.

The next examination given by the American Board of Otolaryngology will be held in Denver, Colorado, at the University Hospital on Monday, September 13, 1926. Application should be made to the Secretary, Dr. H. W. Loeb, 1402 South Grand Boulevard, St. Louis, Missouri.